

ABSTRACT

OF

The Proceedings of the Thirty-Third Annual Meeting of the Association of Life Insurance Medical Directors of America

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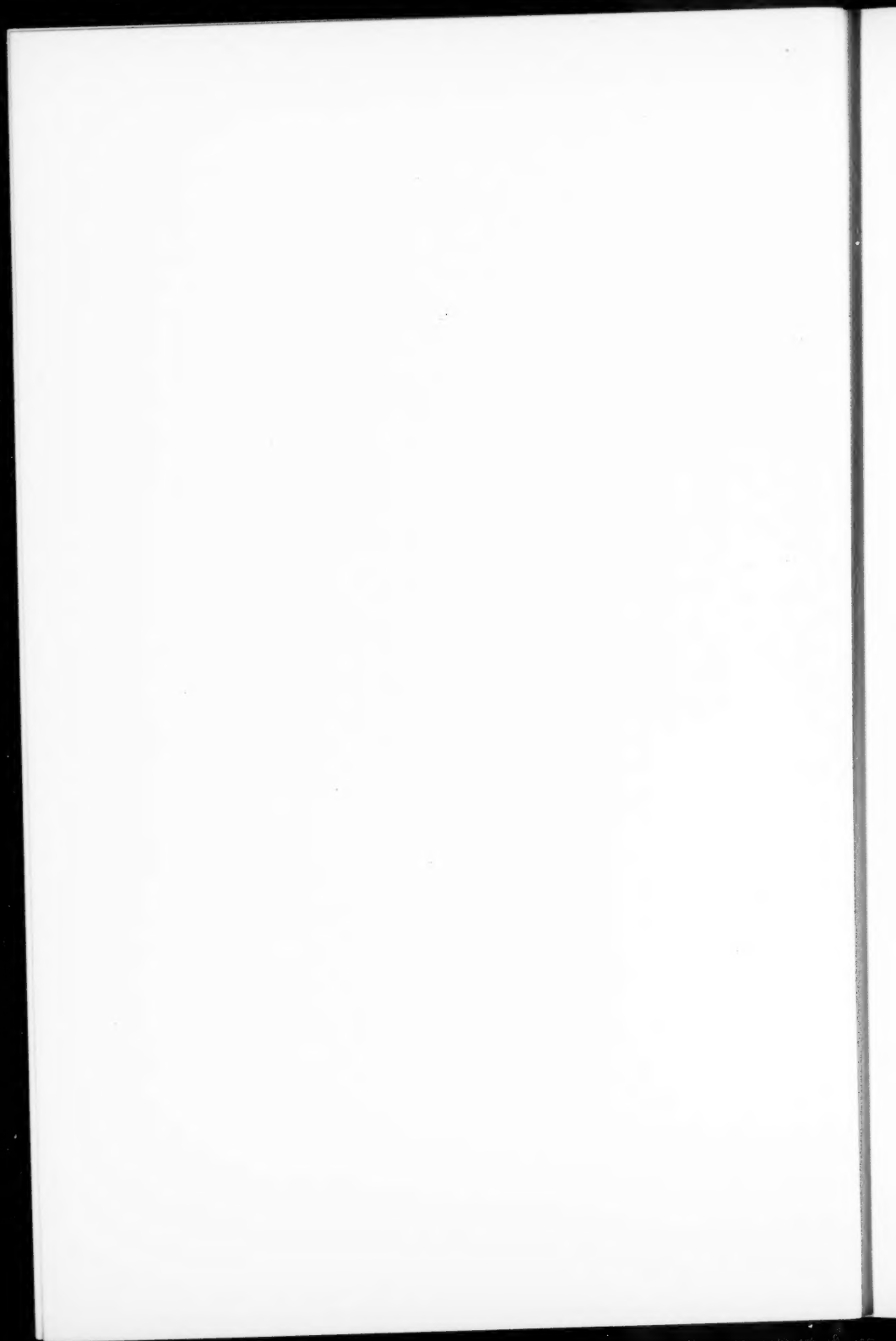
by

THE ASSOCIATION OF LIFE INSURANCE
MEDICAL DIRECTORS OF AMERICA

Compiled by the Editor of the Proceedings

by

★ Order of the Association

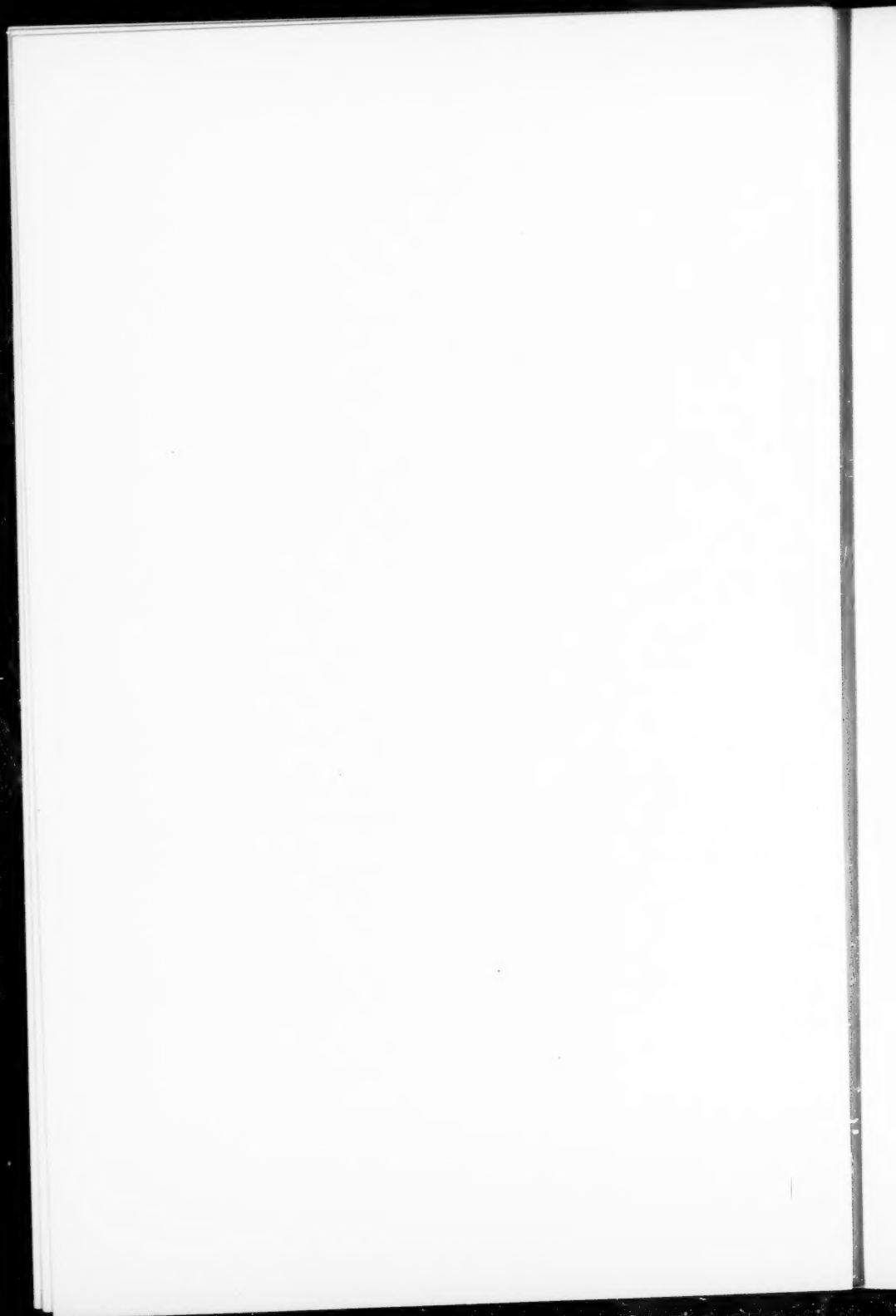


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An Abstract of the Proceedings
OF THE
Association of
Life Insurance Medical Directors
of America

THIRTY-THIRD ANNUAL MEETING

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The Thirty-third Annual Meeting of the Association of Life Insurance Medical Directors of America was held in the Home Office of the New York Life Insurance Company, No. 346 Broadway, New York City, on November 2d and 3rd, 1922. President Thomas F. McMahon was in the chair.

The following members were present at some time during the sessions:

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J. L. Adams, H. B. Anderson, T. D. Archibald, H. A. Baker, A. W. Balch, W. B. Bartlett, J. T. J. Battle, W. W. Beckett, T. W. Bickerton, W. F. Blackford, D. N. Blakely, W. M. Bradshaw, C. T. Brown, W. H. Browne, T. W. Burrows, J. T. Cabaniss, L. D. Chapin, C. L. Christiernin, C. P. Clark, E. A. Colton, H. W. Cook, J. N. Coolidge, G. E. Crawford, R. M. Daley, E. G. Dewis, W. E. Dicker-

man, H. K. Dillard, W. W. Dinsmore, P. G. Drake, E. W. Dwight, O. M. Eakins, Z. H. Ellis, Z. T. Emery, C. H. English, W. G. Exton, J. W. Fisher, Paul FitzGerald, R. A. Fraser, S. W. Gadd, W. S. Gardner, Angus Graham, L. C. Grau, A. H. Griswold, F. L. Grosvenor, G. C. Hall, J. B. Hall, W. F. Hamilton, W. J. Hammer, F. H. Harnden, Whitfield Harral, C. L. Harrison, A. B. Hobbs, J. F. Honsberger, Ross Huston, W. G. Hutchinson, C. B. Irwin, W. A. Jaquith, F. L. B. Jenney, A. O. Jimenis, A. E. Johann, M. L. King, R. J. Kissock, A. S. Knight, W. W. Knight, M. M. Lairy, W. P. Lamb, E. H. Lines, R. L. Lounsberry, L. F. MacKenzie, C. N. McCloud, M. T. McCarty, T. F. McMahon, C. B. McCulloch, O. F. Maxon, Paul Mazzuri, S. W. Means, J. C. Medd, G. L. Megargee, J. H. North, Herbert Old, M. I. Olsen, J. A. Patton, W. O. Pauli, W. A. Peterson, J. S. Phelps, C. B. Piper, J. E. Pollard, W. E. Porter, J. T. Priestley, W. A. Reiter, F. P. Righter, T. H. Rockwell, O. H. Rogers, F. W. Rolph, R. E. Rowley, E. F. Russell, H. C. Scadding, C. E. Schilling, S. B. Scholz, A. L. Sherrill, D. M. Shewbrooks, J. M. Smith, Morton Snow, H. F. Starr, J. B. Steele, Carl Stutsman, Brandreth Symonds, P. E. Tiemann, Harry Toulmin, F. L. Truitt, J. P. Turner, C. A. VanDervoort, Euen Van Kleeck, G. A. Van Wagenen, W. R. Ward, W. H. E. Wehner, F. C. Wells, F. L. Wells, C. F. S. Whitney, T. H. Willard, Gordon Wilson.

There were present also Dr. Kennon Dunham, Dr. H. M. Frost, Dr. S. C. Stanton, and Mr. Arthur Hunter.

Election of New Members

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Total attendance at all sessions, 124.

On motion, the roll-call was waived, and the members were requested to register their names in the book provided for that purpose.

On motion the reading of the minutes of the Special Meeting of the Association, held on December 15, 1921, was waived.

The Secretary read the minutes of the meetings of the Executive Council, of May 8th and November 1, 1922. These minutes were approved as read.

The names of the following candidates for membership in the Association, recommended by the Executive Council, were presented:

Dr. W. F. Milroy, Medical Director, Bankers Reserve Life Company, Omaha, Nebraska.

Dr. Frank Irving Ganot, Medical Supervisor, Prudential Insurance Company of America, Newark, N. J.

Dr. F. L. B. Jenney, Medical Director, Federal Life Insurance Company, Chicago, Illinois.

Dr. Marion Souchon, Medical Director, Pan-American Life Insurance Company, New Orleans, La.

Dr. Henry F. Starr, Medical Director, Southern Life & Trust Co., Greensboro, N. C.

Dr. George W. Parker, Medical Director, Peoria Life Insurance Co., Peoria, Illinois.

Dr. Zenas H. Ellis, Assistant Medical Director, Connecticut General Life Insurance Company, Hartford, Conn.

Dr. Charles Maertz, Assistant Medical Director, Union Central Life Insurance Company, Cincinnati, Ohio.

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Dr. Daniel M. Shewbrooks, Assistant Medical Director, Lincoln National Life Insurance Company, Fort Wayne, Indiana.

Dr. Walter E. Thornton, Assistant Medical Director, Lincoln National Life Insurance Company, Fort Wayne, Indiana.

Dr. Euen Van Kleeck, Assistant Medical Director, Travelers Insurance Company, Hartford, Conn.

Dr. LeRoy C. Grau, Assistant Medical Director, Travelers Insurance Company, Hartford, Conn.

Dr. Walter A. Reiter, Assistant Medical Director, Mutual Benefit Life Insurance Company, Newark, N. J.

Dr. William W. Hitchcock, Medical Director, Occidental Life Insurance Company, Los Angeles, California.

Dr. Elbridge A. Carpenter, Medical Inspector, Penn Mutual Life Insurance Company, Philadelphia, Pa.

Dr. Fred W. Rolph, Associate Medical Director, Confederation Life Association, Toronto, Ontario, Canada.

Dr. Whitfield Harral, Medical Director, Southwestern Life Insurance Company, Dallas, Texas.

Dr. John Mason Little, Ass't. Medical Director, New England Mutual Life Insurance Company, Boston, Mass.

On motion by Dr. Toulmin, seconded by Dr. Willard, the Secretary was instructed to cast a ballot in favor of the election of each of these candidates. The Secretary announced the ballot so cast, and the candidates were declared elected to membership in

the Association. The President appointed Dr. Cook and Dr. Jaquith a Committee to introduce the newly elected members to the Association.

The Secretary announced that the following-named gentlemen were delegates from the American Life Convention Group:

Dr. H. A. Baker, Kansas City Life Insurance Co., Kansas City, Mo.

Dr. M. M. Lairy, LaFayette Life Insurance Company, Cedar Rapids, Iowa.

Dr. G. E. Crawford, Cedar Rapids Life Insurance Company, Cedar Rapids, Iowa.

Dr. Whitfield Harral, Southwestern Life Insurance Company, Dallas, Texas (now elected a regular member of the Association).

Dr. Carl Stutsman, Merchants Life Insurance Co., Des Moines, Iowa.

Dr. W. F. Blackford, Commonwealth Life Insurance Company, Louisville, Ky.

Dr. C. E. Schilling, Ohio State Life Insurance Co., Columbus, O.

Dr. T. W. Burrows, Central Life Assurance Company, Ottawa, Ill. (in place of Dr. Jenney who attends as a regular member of the Association).

Dr. N. T. McCarty, Peoples Life Insurance Company of Indiana, Frankfort, Ind. (in place of Dr. B. C. Brooke, of the Montana Life Insurance Company, who could not attend the meeting).

The following new members and delegates were present and were escorted into the room by the Committee and introduced to the Association: Doctors Harral, Shewbrooks, Rolph, Starr, Jenney, Ganot,

Thirty-Third Annual Meeting

Van Kleeck, Grau, Baker, Crawford, Schilling, McCarty and Stutsman.

Mr. Darwin P. Kingsley, President of the New York Life Insurance Company, welcomed the Association. Mr. Kingsley said:

Mr. Kingsley—Mr. President and Gentlemen: It is a great honor for the New York Life to be the host at this time, and it is a great pleasure for me to say a few words appropriate to the occasion, or as appropriate as I can make them.

I understand this Meeting is likely to be or to become historic. In its membership are new representatives who are here on behalf of that amazing development of life insurance through the West, the South, and the Far West in the United States, and also, as I understand, on behalf of some similar developments in Canada. I do not know how much influence the New York Life had in making the decision that resulted in the membership here to-day. All I know about it is that when Dr. Rogers put the question to me on the theory that he wanted my advice about it, it did not take me long to give him my opinion. That all the records and all the information, on which a scientific and sound selection of lives can be made, should be accessible to every sound and serious and properly conducted life insurance company goes without saying.

The doctor has a theory about this work of yours that is a good deal like the theory I have about the relation of all the English-speaking nations of the world. He thinks there ought to be a federation of all Medical Information Bureaus to include all the Companies of the North American Continent. I think there ought to be a federation of all the peoples in the earth that speak the English language, and when I say a federation I use the word advisedly. I mean the same sort of a binding instrument that was adopted when the thirteen states, independent as they were, were bound together in one nationality. That is what I would do with the English-speaking world. I think that can be done. I think it is practical. I do not know whether the Doctor's idea, on the other

hand, is or not. I hope it can be done. He tells me this morning that he thinks it is going to be done.

At the same time, I know, if I were a Canadian,—(and if I were to be anything else on the face of the earth but an American I would rather be a Canadian)—if I were a Canadian, I would like to go on helping to forget that there is any frontier along that border, but I would be something of a fool if I entirely forgot it, because it is there—unfortunately it is there. And so, if I were arguing to myself as to how far I could go in carrying out Dr. Rogers' idea, I know I would have a little reservation in the back of my mind as to whether I ought not to do something as against that inconceivable day—but a good many inconceivable things have happened in the last eight years—when I would be after all my own master. I sympathize with that feeling. I am sorry to have to, but I know as I say, if I were a Canadian, I would feel that way.

In a sense I am sorry for most of you boys, because I think you are being pushed out of your jobs. I can see what is coming about. You know the statistician and the chemist and the actuary are going to put you out of business. I picked up last night and was reading a part of a lecture by Harry Emerson Fosdick. He is so liberal, you know, that some of the good orthodox people have gotten up recently and shouted that he ought to be put out of the church absolutely. He said, "Picture to yourself what happened in the Twelfth Century in the case of a plague. The churches packed with people, all praying for a miracle by which they might be delivered from the plague. Picture to yourself the same condition in the Twentieth Century. The churches closed absolutely for reasons of health, and everybody would be hunting for the men who had charge of the health regulations of the city." In other words, science has been doing its work and the old order is passing. That is emphatically true in your work. The old day of selecting risks by the whim of the Medical Director and accepting an applicant or not accepting him according to whether the Doctor's breakfast was agreeing

with him, and a lot of other things of that sort, is passing. I suppose, as a matter of fact, that more than sixty per cent of the business of this Company, after it has passed the local Medical Examiners, never goes before a doctor at all. The risk is selected on the facts as they are. It is not a matter of opinion or guess. It is action based on statistics. Some of the doctors I know in our own Medical Department are a little sore because they say the whole medical shop is run by the Actuary. Well, I suppose it is. How far we shall go ultimately I do not know. Of course, we shall never get to the point where when a case is irregular the Medical Director will not have to be consulted, but we are passing rapidly out of the old method and the Medical Director in the old sense of it, before very long, will be as dead as the dodo bird. That is the modern way of doing things. In fact, the medical work of life insurance companies has been for many years the one unscientific thing in life insurance selection. Life insurance itself is scientific. It is based on scientific processes and scientific ideas, exact ideas. There should be no guessing about it, but the medical part of the work has been only semi-scientific until recent times. The medical part of the work will steadily become more and more scientific.

The New York Life is glad to have you here. We are especially glad because, assuming that there is virtue or fault to be attached to the newer methods of selecting risks, the New York Life is entitled to some credit if the method is sound, and if it is at all unsound we are prepared to meet that too. We believe this work will go on. How far, of course, no one can say. We are committed to it irrevocably, and we are prepared to share our knowledge with anybody and everybody engaged in this beneficent enterprise.

Mr. President, in behalf of the New York Life, and especially in behalf of its Medical Staff I welcome you to this Meeting. I hope that it will ultimately reach the point of which Dr. Rogers dreams, of a federation of all the sound, solid, respectable and reputable life insurance companies of the North American Continent.

Dr. T. F. McMahon, President of the Association, delivered the following address:

Allow me first to express my appreciation of the honor you have done me in electing me president of our Association.

I am fully conscious that my election is not due to any merit of my own, but is the expression of your desire to show your friendship for your Canadian neighbors.

For one hundred and ten years we have lived in peace and harmony and our regard for one another is growing steadily as the years pass by. Time and again you have expressed your appreciation of the spirit of our young dominion in sending over half a million soldiers to Europe, and we in turn recognize that the vast and magnificent army and the financial aid contributed by your great republic made victory certain for the allied cause and saved the world from chaos and despair.

For the past two years a Committee of Actuaries and Medical Directors of the Canadian Companies has met weekly except during the summer months to study substandard lives and to endeavor to bring about harmony in our treatment of various impairments. The report to be presented by Dr. Scadding and Dr. Strathy will give you a synopsis of the results of our labors. We have found those meetings most profitable. The Actuaries and Medical Directors understand one another better and companies are acting in greater harmony than ever before. We are grateful to Mr. Arthur Hunter and Dr. Patton for coming to Toronto to assist us with their great experience and excellent advice. I venture to suggest that in many districts it would be possible to hold similar meetings and that the result would be a vastly increased ability to deal with insurance problems.

I have another suggestion to make for your consideration. The President for the year is responsible for the program of the Annual Meeting. This seems to me to be a somewhat haphazard system. Some subjects may receive an undue amount of attention and others of greater importance may meet with

comparative neglect. Would it not be possible for a small committee to be appointed whose duty will be to block out a plan of campaign for some years in advance so that men specially fitted for the study of certain impairments may be selected and have ample time to prepare their contributions, and so that no subject of importance will be neglected. I hope also that some future competent and not too modest President will devote his annual address to advice to the younger Medical Directors in the performance of their duties. You will all appreciate how valuable an address of this kind from such men as Dr. Symonds and Dr. Rogers would be.

There are yet many subjects on which the average Medical Director needs light. In the program for this year I have endeavored to get some light on two subjects of some importance, namely, epilepsy and focal infections. It is not right in my opinion to deny the epileptic insurance on some terms. Surely it is possible to arrive at some safe and equitable basis for insuring many of these applicants. We also need light on cases where the applicant shows a history of mental derangement many years ago but who now appears to be permanently cured.

Syphilis.—In the whole range of impairments there is no subject that has given me more concern than syphilis. To decline every syphilitic is a simple solution of the problem, but it is not an equitable one. The subject is one of tremendous importance from the life insurance standpoint. The New York Health Department has estimated that about 8% of the civilian population is infected with syphilis and gonorrhoea is still more common. The Canadian National Council for combating venereal diseases estimates that in Canada we have over half a million of cases of syphilis alone. The conditions in Great Britain and France are much worse. An active campaign is being carried on for combating these diseases. Have the Life Insurance Companies done their share? Would it not be good business for the Companies to give financial aid to this campaign. The mortality from tuberculosis has been greatly reduced by the measures taken for preventing and combating

this disease. Is it not possible to secure similar results in the prevention and cure of syphilis? The mortality direct and indirect, from this disease must be appalling. The death certificates for obvious reasons do not give the facts. Some applicants for insurance give a history of syphilis, many more in my opinion suppress that history. It follows that we are insuring many syphilitics without the rating that this disease calls for. Can anything be done to detect a larger proportion of these cases? Is it feasible to have the Wassermann reaction in a considerable number of cases where high blood pressure or other symptoms make us suspicious? Or should our energies be directed to the prevention and early treatment of the disease on a large scale? Can we secure more guidance in our selection of risks than we have at present? How shall we deal with applicants who show a history of syphilis thoroughly treated five, ten or fifteen years ago? Or with applicants who in spite of a history of syphilis not thoroughly treated have enjoyed many years of excellent health and show no indication of the disease? What dependence is to be placed on the Wassermann test in our selection? These and many other problems face us and cry for solution. Would not a symposium on syphilis at some future meeting be profitable if the incoming President selected the men to deal with the subject now and give them ample time to do their work well?

Insurance Without Medical Examination.—This subject has excited a good deal of interest in Canada during the past two or three years. In the time at my disposal I can only touch the fringe of the subject and I would refer those interested to the papers by Mr. Reid of the London Life, Mr. Kilgour of the North American Life, and Mr. Arthur Hunter of the New York Life and the excellent discussion of these papers published in the transactions of the Actuarial Society of America, 1921. I think the time has come for Medical Directors to take part in this discussion.

In my Company the non-medical business issued from January 1, 1921, to September 1, 1922, shows 8463 policies for \$9,148,000.00. In 1921 the ratio of policies issued in Canada

without medical examination was 32.31% in the number of policies and 15.3% in the amount of insurance. In 1922 to October 1st the ratio was 46.18% in the number of policies and 24.78 in their amount. The increase in the percentage in 1922 was caused largely by increasing the limit of non-medical policies from \$1000 to \$1500. This increase is more apparent in the later months. In September the ratio was 55.9% in the number and 35.4% in the amount. These figures show that nearly half our policies in Canada are issued without medical examination.

Our medical examiners, therefore, receive approximately only one half the fees they would if an examination were called for in every instance. If it is true that one of the chief causes for adopting non-medical insurance by so many Companies in Canada was the badly advised agitation by some of the Medical Societies to bring about a large increase in the fees, it would appear that they have succeeded in half killing the goose that lays the golden egg. Some of the demands made by medical societies were impossible, if not ridiculous.

The actuaries are divided in their attitude toward non-medical insurance and the doctors have not yet discussed the subject or expressed their opinions on the matter. Naturally enough the medical examiner is inclined to question closely any project which involves the selection of risks without examination. He is apt to assume that medical selection has advantages that are not entirely supplanted by a selection partly based upon reports by agents and reporting companies.

A large proportion of our claims are the result of cardio-renal-vascular diseases. It is fair to assume that medical selection will lead to keep this class within normal bounds. In this way we may hope to eliminate at least in part:

- (1) Those who are medically unfit and knowing it seek to take advantage of the non-medical selection;
- (2) Those who are medically unfit without their own knowledge; and,
- (3) Those who suffer from impairments which make them decidedly substandard.

Amongst the other conditions which might be detected by a medical examination but escape other methods I might mention:

1. Incipient or arrested tuberculosis.
2. Cancer and other malignant growths.
3. Diseases of the gall bladder, gastric or duodenal ulcer, and chronic appendicitis.
4. Diabetes and glycosuria.
5. Goiter including exophthalmic goiter.
6. Various diseases of the nervous system, including paresis, locomotor ataxia, paralysis and milder degrees of mental impairment.
7. Diseases of the bladder and urethra.
8. Anæmia, including the pernicious type.
9. Syphilis with visible or other detectable lesions.

Dr. Symonds points out that:

"There is a distinct correlation between the percentage of risks 'Disapproved by the Medical Examiner' and the mortality of the first and subsequent policy years. This correlation is negative and, therefore, the mortality becomes smaller as the percentage of 'Disapproved by the Medical Examiner' becomes larger. This correlation shows some evidence even up to the tenth policy year."

On the other hand there is some manifest advantage in accepting applications for small policies without medical examination. The agents will certainly secure a larger number of policies, thus extending the benefits of life insurance to a larger number of people. The difficulty which the agents have met with, especially in remote districts, in getting prompt medical service will be eliminated. In addition to this we must not forget that the saving of medical fees will reach a large sum.

In the United States the question has not yet become a live one but there can be little doubt that it will soon be knocking

at your doors. I would, therefore, advise you to take time by the forelock and study the problem so that you may be in a position to express your opinion whether it is a forward movement worthy of encouragement or a change that carries with it dangers that must be carefully considered.

In conclusion I would like to express my appreciation of the generous response made by the members whom I invited to contribute to the program. I am afraid I have overlooked some of the younger men who would have added to the value of the meeting, but this is because I do not know them well enough. I made an honest effort to secure some contributors but their modesty prevented them from responding. I would like also to return my thanks to the Secretary, Dr. Hobbs, for the help I received from him, without which I should have had a sad time.

Dr. McMahon—It is with deep regret that I announce the death during the past year of two of the members of this Association—Dr. Thomas C. Craig and Dr. Greenly V. Woollen. Dr. Z. Taylor Emery has prepared a memorial on Dr. Craig and Dr. J. M. Smith has prepared a memorial on Dr. Woollen which will be read by the Secretary.

The Secretary read the following memorials:

Thomas Canby Craig, U. S. N. retired, Asst. Medical Director of the Manhattan Life Ins. Co. for over 20 years, died on the 13th of December, 1921, at his home 275 Clinton Ave., Brooklyn, N. Y., of angina pectoris, due to arteriosclerosis.

Dr. Craig was born near Chambersburg, Pa., and at the time of his death was 68 years of age. He was educated in the school of Chambersburg, and also graduated from the Medical Department of the University of Pennsylvania in 1880, served a year as surgeon of the Blocksley Hospital, and then entered the Navy as a Navy Surgeon, remaining in active

Memorials of Dr. Craig and Dr. Woollen 15

duty for 18 years. He was retired for physical disability, and remained on the inactive list, except for a few months during the Spanish-American War, until the time of his death. He served in the Far East, Egypt, and South America, was in the navy during the Hawaiian rebellion, and served in the Spanish-American War. He had travelled around the world three times, and upon his retirement he came to Brooklyn, where he practiced his profession since 1898. At the time of his death, and for many years prior thereto, he was a deacon of the Lafayette Avenue Presbyterian Church. He took an extended course in pathology and bacteriology, under the late Dr. E. H. Wilson, Medical Director of the Hoagland Laboratory, and at that time he did a large amount of laboratory work in connection with the cause of diseases, particularly in connection with the work of the Board of Health, and he showed a high degree of technical skill. He served, for some years, on the staff of the Brooklyn Hospital for Consumptives. He wrote many articles on Diagnostic and laboratory work and was regarded as an expert in this line by the profession of Brooklyn. He was a member of the Medical Society of the County of Kings, the New York State Medical Society, the American Medical Association, the University Club of Brooklyn, and the Navy Athletic Club. During his long connection with the Manhattan Life Ins. Co., he exhibited a high degree of skill, in the examination of and the selection of risks, and was highly regarded by his associates. Through his death, the profession, at large, lost a distinct asset.

W. TAYLOR EMERY, M. D.
Medical Director.

Dr. Greenly V. Woollen, age 81 years, Medical Director of the American Central Life Insurance Company, died at his home in Indianapolis, Indiana, December 10, 1921. He was born in Indiana, June 24, 1840, and graduated from the Bellevue Hospital Medical College in 1865. He was one of the founders of the Indiana State Medical Association, and

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served as its secretary from 1865 to 1875. Later, he served as the president of the Indianapolis Medical Society. He held the chair of Professor of Rhinology and Laryngology in the old Central College of Physicians and Surgeons, Indianapolis, until he gave up his specialty to become Medical Director of the American Central Life Insurance Company. Dr. Woollen was the first superintendent of the City Hospital of Indianapolis, and served as visiting physician to the hospital for twenty-five years. During the Civil War, which began in his twenty-first year, he served as Army Surgeon from September, 1861, to September, 1864.

At the close of the war, he began the practice of general medicine, and in 1884 took up the special study of diseases of upper air passages, taking an extended course in this branch at the New York Polyclinic Post Graduate School and later in the London Throat Hospital as a protégé of Sir Morell McKenzie. During his years of active practice, he was a frequent contributor of papers on general medicine published in many medical journals.

As a citizen, Dr. Woollen stood exponent of liberality and distinctive public spirit. For many years he was chairman of the Board of Directors of Franklin College, also of Crawford Industrial School, Zionville, Indiana, as well as deacon of the First Baptist Church of Indianapolis. He was interested in the basic art of agriculture and allied industries and was a member of the Indiana Jersey Cattle Club and Indiana Dairy Association. He was also a member of the G. H. Thomas Post No. 17, G. A. R., Military Order of Loyal Legion of U. S., and of the Indianapolis Commercial and Columbia Clubs.

While he was a physician of the Old School, he kept in advance of his contemporaries and stood high in the specialty to which he devoted the best years of his active practice, and in later life, he found congenial employment in his work as Medical Director of the American Central Life Insurance Company, in which capacity he served with loyal devotion for sixteen years.

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On motion, these memorials were ordered spread upon the minutes of the Association.

Before taking up the report of the Nominating Committee, it was moved by Dr. Toulmin and seconded by Dr. Willard that Article XIII of the Constitution be waived. The motion was carried. Article XIII of the Constitution reads as follows:

"AMENDMENTS. This constitution may be amended by a vote of two-thirds of the members present at any meeting held at least three months after notice of any proposed amendment shall have been sent to each member by the Secretary."

It was moved by Dr. Toulmin and seconded by Dr. Willard that Article V of the Constitution which now reads—

"EXECUTIVE COUNCIL. The Officers of the Association, former Presidents and three other members, not officers, shall constitute the Executive Council,"

be amended to read—

"EXECUTIVE COUNCIL. The Officers of the Association, former Presidents and five other members, not officers, shall constitute the Executive Council."

The motion was carried.

The Nominating Committee then presented the following report:

November 1, 1922.

The Nominating Committee begs leave to make the following report:

At a meeting held November 2, 1922, the Committee by votes duly seconded and carried decided to recommend the nominations of the following:

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For <i>President</i>	Dr. FRANK L. GROSVENOR of the Travelers, Hartford, Con- necticut.
" <i>First Vice-President</i>	Dr. WILLIAM R. WARD of the Mutual Benefit, Newark.
" <i>Second Vice-President</i>	Dr. CHESTER F. S. WHITNEY of the Home Life, New York.
" <i>Secretary</i>	Dr. ANGIER B. HOBBS of the New York Life.
" <i>Treasurer</i>	Dr. CHARLES L. CHRISTIERNIN of the Metropolitan Life.
" <i>Editor of the Proceedings</i>	Dr. ROBERT M. DALEY of the Equitable Life.
" <i>Members of the Executive Council</i>	Dr. GEORGE A. VAN WAGENEN of the Mutual Benefit Life, Newark. Dr. EDWIN W. DWIGHT of the New England Mutual, Boston. Dr. J. ALLEN PATTON of the Prudential, Newark. Dr. F. L. B. JENNEY of the Federal Life Insurance Com- pany, Chicago. Dr. W. W. BECKETT of the Pacific Mutual, Los Angeles.

All of which is respectfully submitted.

WM. EVELYN PORTER, M.D.
CHESTER T. BROWN, M.D.
HARRY TOULMIN, M.D.
C. F. S. WHITNEY, M.D.
ROBERT M. DALEY, M.D.
THOMAS H. WILLARD, M.D.—*Chairman.*

Dr. McMahon—You have heard the report of the
Nominating Committee. Has any member any
further nominations to present?

Report of Committee on Public Health 19

No further nominations being presented, it was moved by Dr. Jaquith and seconded by Dr. Rogers that the nominations be declared closed and the Secretary instructed to cast a ballot on the morning following in favor of the election of the Officers and members of the Executive Council so nominated. The motion was carried.

The Treasurer read his report. The Auditing Committee appointed by the Chair, Drs. Grosvenor, Jaquith and Ward, reported that they had reviewed the books of the Treasurer and audited his report and found the same to be correct. The report of the Treasurer was accepted and ordered placed on file.

Dr. Thomas H. Willard, Chairman of the Committee on Public Health, reported as follows:

"Mr. President: There is no formal report by the Committee on Public Health. As set forth in our report made last year, the Public Health Committee of the Association of Life Insurance Medical Directors is marking time, and is holding itself in readiness to coöperate with the Association of Life Insurance Presidents, whenever, wherever and however they in their wisdom may see the advantage of calling upon us for assistance. Nothing of a serious matter has come up to call for activity on the part of the Committee. As a Committee we have kept throughout the year in constant and close conference with the people representing the Association of Life Insurance Presidents."

On motion, the report of the Committee of Public Health was accepted and placed on file.

Dr. Rogers, Chairman of the Committee appointed in 1921 to study the work of Dreier in relation to Life Insurance, made the following report:

"Mr. President: I have an apology to make to the members of this Association. During the past year, my time was so taken up with the work of admitting the A. L. C. Companies and with other matters, and because of my absence from the city for two months, there has been no meeting of the Committee, but the members of the Committee have been engaged in collecting data, notably Dr. Symonds and Dr. Dublin of the Metropolitan, and there have been some data accumulated in the office of the New York Life. The Committee has estimated that, to make a thorough investigation of the subject will probably cost as a minimum, \$10,000, and while the Committee has had no formal meetings, it has been in conference and believes that if the Association will indulge it and permit it to run over to next year, it will be possible at that time to lay before the Association a report that will give the members of the Association a clear idea of what the problem really amounts to. I may say that one fact has been clearly brought out in the figures that have been thus far accumulated, and that is, that the height of an individual is curiously disproportionately distributed in a good many cases, and that, when you have the height and weight of a man, you are not getting any more than a provisional idea of the man's build. It may very well be that some day we shall find that the stem length or the spine length will be the proper measure to take as a basis for estimating the value of a risk, but it involves a fundamental change in our methods, it involves the overthrow of all of our statistics with reference to build, and it is really a very formidable subject.

"In conclusion I should like to say a personal word, and that is that I have been in one way or another tangled up in so many different kinds of matters that I should appreciate very much if the Association would indulge me by allowing me to withdraw from the Chairmanship of this Committee. I think that Dr. Symonds has shown such peculiar ability for this kind of work, that it would be a valuable move for the Association if you would allow me to withdraw from the Chairmanship of the Committee, besides being a very kindly

Report of Committee on Standardization 21

thing to do to me. I want to serve on the Committee with Dr. Symonds in charge as Chairman."

Dr. Rockwell—As a member of the Committee, I would endorse Dr. Rogers' request.

Dr. Symonds—I personally do not see the need of making any change. I am perfectly willing to do the work under Dr. Rogers as Chairman.

Dr. Willard—I move that the report be referred back to the same Committee, with Dr. Symonds as Chairman instead of Dr. Rogers. Dr. Toulmin seconded the motion and it was carried.

Dr. Symonds, Chairman of the Committee on the Standardization of the Medical Blank, made the following report:

"Mr. President: The Committee on the Standardization of the Medical Blank took up this question which was submitted a year ago. As Chairman of the Committee I consulted most of the Companies in New York City; and Dr. Toulmin conferred with those in Philadelphia. The nearly unanimous opinion was that in neither New York nor Philadelphia did the Companies have any use for a standard blank. All of the Companies have the blanks standardized to a certain size, and the equipment has been made to conform to those requirements. Our hosts, for instance, would not think for a moment of giving up their little half sheet, nor would the Metropolitan dream of giving up their blanket, at the present time. We have in our own Company more than a half million applications in the current files, and these are being added to at the rate of one hundred and twenty-five thousand a year, and perhaps seventy-five thousand are abstracted, but we have our equipment for that sized blank, and the same thing will apply to all the other large Companies, so much so, that I have received no affirmative responses to my query as to the standardization of the medical blank.

"One of the members of the Committee, Dr. Cook, made a

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very happy suggestion and that was that, while the blank perhaps could not be standardized in view of the physical requirements, yet on the other hand the questions in the blank could be reviewed and, to a certain degree standardized, by a continuing Committee to whom the questions might be referred, as to whether they were proper questions, or whether certain omitted questions should be inserted and the Committee is unanimously of the opinion that a continuing Committee on the standardization of the questions, or perhaps, to put it without the word standardization, a Committee to suggest and to receive suggestions and to review the questions on the Medical Examination Blanks, should be appointed."

On motion the report of the Committee was accepted and placed on file and the President announced that the same Committee would be continued to study the questions on the medical blanks.

Dr. McMahon—It has been thought best to take up the report of the Blood Pressure Committee in connection with the discussion of Dr. Symonds' paper, entitled "The Blood Pressure of Healthy Men and Women" which will now be taken up for discussion.

THE BLOOD PRESSURE OF HEALTHY MEN AND WOMEN

BY BRANDRETH SYMONDS, M.A., M.D., LL.D.

Chief Medical Director, Mutual Life Insurance Co., of New York

SYSTOLIC PRESSURE—MEN

This study is based upon the record of risks accepted at standard rates by the Mutual Life Insurance Company for the years 1907 to 1919 both inclusive. More than 95% of the

readings were taken by our New York City examiners and our medical referees and their immediate assistants. In the earlier years no readings were taken except by these examiners but in the later years a few reports were made by our other examiners in the field. These reports amounted to less than 6% in the year 1919. In the earlier years most of the readings were obtained by palpation, but since 1915 nearly all of them have been taken by auscultation. The readings were taken while the applicant was seated and the apparatus was about the level of his heart. A wide cuff was used and in most cases the Tycos manometer, devised by our former President, Dr. Rogers.

It is well known that the systolic pressure increases with age and with weight and also that weight increases with age. In order to reach proper conclusions regarding these three variables, age, weight and pressure, it is necessary to separate each age into weight-groups. For convenience our material has been arranged according to the Build-groups of the Medico-Actuarial Mortality Investigation (1). The basis of these groups is the average weight for each inch of height at age 37. Build-group 0 comprises those within 5% above and below this standard. Build-group 1 comprises those from 5% to 15% above the standard; Build-group 2 15% to 25% above the standard; Build-group 3 25% to 35% above; Build-group 4 35% to 50% above; and Build-group 5 those more than 50% above the standard. The under-weights start with Build-group 6 which comprises those who are 5% to 15% less than the standard; Build-group 7 15% to 25% below standard; Build-group 8 25% to 35% and Build-group 9 those who are more than 35% below the standard.

The following abridged table of the Build-groups at the heights, 5 ft. 4 in., 5 ft. 8 in., and 6 ft. will refresh our memories as to the general range of the Build-groups at the other heights.

The average systolic pressures among healthy men when analyzed according to age and build-group are set forth in Table 2.

TABLE I

BUILD-GROUPS

	(9)	(8)	(7)	(6)	(5)	(4)	(3)	(2)	(1)	(5)
5 ft. 4 in.	90 & Under	91-104	105-118	119-132	133-147	148-161	162-175	176-189	190-210	211 & Over
5 ft. 8 in.	101 & Under	102-116	117-132	133-148	149-164	165-180	181-196	197-211	212-235	236 & Over
9 ft. 0 in.	115 & Under	116-133	134-151	152-169	170-188	189-206	207-224	225-242	243-268	269 & Over

TABLE 2

SYSTOLIC PRESSURE—MEN ONLY

Age	Bld. 9		Bld. 8		Bld. 7		Bld. 6		Bld. 5		Bld. 4		Bld. 3		Bld. 2		Bld. 1		Bld. 0		Bld. 1		Bld. 2		Bld. 3		Bld. 4		Bld. 5		Total	
Periods	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.
15-19	114.3	3	113.6	259	119.1	2,300	121.9	3,227	123.4	1,197	127.4	197	125.8	63	131.9	13	120.0	4	121.2	7,263												
20-24	121.0	1	120.1	173	121.2	3,070	123.3	9,102	124.3	5,781	125.8	1,010	125.8	444	127.2	130	122.4	23	123.0	1	123.4	20,904										
25-29			119.8	142	122.0	3,539	122.9	10,061	124.4	8,603	126.2	3,540	126.6	1,459	125.7	341	128.1	63	123.0	1	123.9	27,737										
30-34			120.9	95	121.0	2,639	122.4	7,833	123.9	9,381	125.0	5,089	126.2	2,180	127.7	595	128.6	118	130.0	1	123.8	20,592										
35-39			120.8	52	122.3	1,133	123.3	3,868	126.3	5,506	127.7	4,211	129.2	2,525	130.9	811	131.7	162	130.0	1	126.5	18,142										
40-44			116.7	31	124.9	661	125.8	2,401	127.4	3,833	129.7	3,332	130.8	1,988	132.7	600	133.6	113	133.0	4	128.4	12,063										
45-49			122.5	19	125.9	325	128.3	1,260	129.8	2,236	132.5	1,902	133.3	1,226	133.5	384	133.2	76	131.5	2	130.9	7,430										
50-54			128.8	12	129.1	196	131.0	618	133.1	1,034	134.8	978	137.5	646	136.1	188	136.9	19			130.9	3,691										
55-59			124.4	5	133.4	111	132.8	348	135.1	601	136.2	489	137.7	205	135.9	65	135.2	5			135.2	1,889										
60 & Ov.																																
All Ages	114.2	5	118.3	844	121.6	16,327	123.5	44,445	125.4	44,425	127.8	26,300	129.1	13,407	130.2	3,896	130.6	786	131.0	14	125.3	150,419										

This table should be read in connection with Table 1. If a man is 5 ft. 8 in. tall and weighs 150 pounds, we expect him to show a systolic pressure of 123.4 mm. when he is 18 years old; of 123.9 mm. when he is 32 years old; and of 129.8 mm. when he is 53 years old. Similarly if a man is 37 years old and 5 ft. 8 in. tall, we expect him to show a systolic pressure of 119.8 mm. if he weighs 112 pounds; of 124.4 mm. if he weighs 160 pounds; and of 129.3 mm. if he weighs 205 pounds.

Table 2 is based upon 150,419 entrants, men only. Even with this there are some irregularities where the data are scanty. Perhaps there are also evidences of increasingly greater care in the selection of the over-weights as in Build-groups 4 and 5, for these do not show the same proportionate increase in pressure as the other build-groups. On the other hand it is possible that in these marked over-weights some of the cases are already experiencing a certain amount of interference with the heart-action on account of the fat.

In looking across the table it will be noticed that there is an increase of about 11 mm. between very light-weight group 8 and very heavy-weight group 4 in each age-period. Also if we look down each Build-group it will be noticed that the pressure increases about 11 mm. between the youngest and the oldest. Nearly all of this increase comes after 40 for 2 mm. will cover the increase up to that age in most of the build-groups.

If we take into account all the factors, especially with regard to the youthful light-weights and the marked over-weights, Table 3 is submitted as representing the systolic pressure which may be expected "on the average," for the corresponding age-period and build-group in reasonably healthy men in the United States and Canada, unless marked leanness or fatness be considered a sign of ill health. The last column, headed "All Builds" and the last row, designated "All Ages," represent the average of the preceding figures, as if each group contained the same number of entrants. In Table 2 the corresponding averages are affected by the fact that the entrants in different groups vary. Thus an item in the low average of ages 15-19 and 20-24 in Table 2 is the fact that three-quarters

of their entrants are in the lighter build-group. Also the low averages of Build-groups 8 and 7 in Table 2 are partly because so many of their entrants are in the younger ages.

TABLE 3
THEORETICAL SYSTOLIC PRESSURE FOR MEN
Build-Groups

Ages	(9)	(8)	(7)	(6)	(5)	(4)	(3)	(2)	(1)	(0)	All Blds.
15-19	114	116	120	122	123	125	126	128	130	131	123.5
20-24	117	119	121	123	123	125	126	128	130	131	124.3
25-29	117	120	121	123	124	125	126	128	130	131	124.5
30-34	118	120	121	123	124	126	127	129	131	132	125.1
35-39	118	121	122	123	124	126	127	129	131	132	125.3
40-44	119	121	123	124	126	127	129	130	132	133	126.4
45-49	121	122	125	126	127	129	131	132	134	135	128.2
50-54	123	124	126	128	130	131	133	134	136	137	130.2
55-59	126	128	129	131	133	134	137	138	139	140	133.5
60 & Ov.	128	129	133	133	135	136	138	139	140	142	135.3
All Ages	120.1	122.0	124.1	125.6	126.9	128.4	130.0	131.5	133.3	134.4	127.6

It is not known what is the range above and below these averages which will give reasonable mortalities. The mortality-ratios have been calculated but the exposure is short and is complicated by the fact that it covers two of the years of the epidemic influenza. As a result of this many of the deaths occurred in the ages below 30 and therefore in the groups with low blood-pressure. Dr. Fisher (11) has given us most valuable information on the effect of high pressure and has promised to make another contribution at this meeting.

One point is of interest however. If we eliminate those risks whose pressure is above 140 mm., we get a very curious result which is set forth in Table 4. This shows the average systolic pressure of those left after the eliminations and the percentage of the risks eliminated from each group. Build-groups 9 and 5 have been omitted because no risks were eliminated from them.

If we compare this with Table 2 we will see that we have eliminated only 8,579, or 5.7% and still have 141,840 entrants. The general average for all ages and all builds has been reduced only 1.3 mm. The averages for all the older ages and all the

TABLE 4
SYSTOLIC PRESSURE—MEN (140 MM. AND BELOW)

Age Pres- sure	Bld. 8		Bld. 7		Bld. 6		Bld. 5		Bld. 4		Bld. 3		Bld. 2		Bld. 1		Bld. 0		Total	
	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated
15-19	113.6		118.0	7.7	121.8	8	123.0	1.7	126.9	3.0	130.6	7.7	125.1	3.2	126.9	3.0	123.0	1.7	121.6	1.0
20-24	119.4	6	120.6	1.3	122.5	1.6	123.8	2.4	125.3	3.6	127.5	4.3	124.3	4.8	126.9	3.0	123.0	1.7	123.0	1.0
25-29	120.2	1.4	121.7	1.3	122.5	1.9	123.8	2.4	125.3	3.5	127.5	4.3	124.3	4.8	126.9	3.0	123.0	1.7	123.0	1.0
30-34	120.2	3.2	120.7	1.4	121.9	1.9	123.3	2.9	124.8	3.9	126.0	8.7	124.9	5.8	126.0	8.7	126.0	11.0	123.1	3.1
35-39	110.8		121.4	2.2	122.6	2.8	123.6	3.9	125.5	5.8	127.5	10.1	125.8	7.9	127.5	10.1	126.3	11.6	123.9	4.6
40-44	120.4	1.9	121.5	3.3	123.1	5.1	125.0	5.6	126.0	8.7	127.5	13.3	127.2	11.0	127.5	13.3	130.2	9.9	125.0	7.2
45-49	114.3	6.5	123.5	6.1	124.1	7.7	125.3	9.6	127.0	13.9	127.7	15.8	127.7	15.8	129.2	20.5	129.2	26.6	125.9	11.8
50-54	121.3	5.3	124.2	7.7	125.7	12.5	127.1	13.7	129.1	19.5	129.9	23.3	129.9	23.3	129.9	21.9	128.2	26.3	127.7	16.8
55-59	128.8		127.0	10.2	127.4	18.6	128.9	21.3	130.9	25.6	131.3	29.8	131.3	31.6	131.3	29.8	133.9	26.3	129.7	23.9
60 & Ov.	124.4		129.2	25.2	128.0	24.7	129.0	31.1	130.8	31.7	130.0	35.4	131.8	37.4	130.0	35.4	128.0	40.0	129.8	30.7
All Ages	117.9	1.2	121.1	2.1	122.7	3.2	124.2	5.2	126.1	8.6	127.6	14.2	126.8	11.9	127.6	14.2	127.9	14.8	124.9	5.7
Entrants	834		15,991		43,034		42,133		24,036		3,344		11,805		644		141,840			

heavier build-groups have been markedly lowered. The difference between the youngest and oldest age period is only 8.8 mm. instead of 13 mm. as before. The marked increase at age 40 has also disappeared or at least has been postponed until age 55. Looking across the table the differences between the build-groups are not as great as before. Evidently a large part of the differences in Table 2 is due to the inclusion of blood-pressure above 140 mm. But there are only 704 entrants whose pressure is above 150 mm. and only 372 of these whose pressure is above 155 mm. Practically all of those eliminated lay between 141 mm. and 150 mm. both inclusive. The larger averages therefore in the older ages and in the heavier build-groups are due to the inclusion of an increasingly larger number of risks whose blood-pressures are not unusually high. It is tempting to regard any systolic pressure above 140 mm. with suspicion but this view has not yet been supported by any mortality reports. Rogers & Hunter (2) though make a charge for a systolic pressure of 140 mm. up to and including age 35, according to the system of numerical rating which is in vogue in the New York Life.

The material as a whole represents carefully selected, healthy risks. Those issues of 1907 to 1917, which form part of Table 2, when exposed to 1918 showed 1,367 actual deaths, and 2,095.5 expected deaths calculated by the Medico-Actuarial Mortality Table, which gives a mortality ratio of 65.2%. This is 9.6 points less than the general mortality of the Mutual Life for the same period and issue and is due to the superior quality of the medical examiners who made the reports on blood-pressure, for more than 95 % were made by our New York City men and our medical referees and their immediate assistants.

Life insurance has furnished the only large collections of reports on the blood-pressure of healthy men. A few articles have appeared on the blood-pressure of students (3), (4) and of soldiers (5), (6) but the ages have been limited to those comparatively young. Four excellent reports have been made from life insurance data. One was made by Dr. J. W. Fisher

(7), based on records of the Northwestern Mutual Life Insurance Company and the Mutual Benefit Life Insurance Company. The number of entrants in the ages below 36 is rather small but above that age the cases are numerous. The report was made in 1914.

A very extensive report was made by Dr. Mackenzie (8) to our association at its 26th annual meeting in 1915, based upon examinations made in the previous three years. The cases below age 50 were numerous.

Another report was made by Hunter & Rogers (9). The observations were made during 1913 to 1916, both inclusive and all of the cases were accepted on standard plans. The entrants were numerous.

Another excellent report was made by Goepp (10) on cases issued by the Provident Life and Trust Company during 1918. The cases below age 48 were numerous.

A résumé of the results obtained by these four reporters is set forth in Table 5.

TABLE 5

FISHER		MACKENZIE		HUNTER & ROGERS		GOEPP	
Ages	S. P. B.	Ages	S. P. B.	Ages	S. P. B.	Ages	S. P. B.
15-20	119.85	15-19	119	15-19	120		
21-25	122.76	20-24	122	20-24	122	22	119.5
26-30	123.65	25-29	123	25-29	123	23-27	120.8
31-35	123.74	30-34	124	30-34	124	28-32	122.4
36-40	126.96	35-39	126	35-39	125	33-37	122.6
41-45	128.56	40-44	127	40-44	127	38-42	124.2
46-50	130.57	45-49	129	45-49	129	43-47	125.8
51-55	132.13	50-54	132	50-54	133	48-52	128.6
56-60	137.78	55-59	135	55-59	134	53-57	129.4
		60-64	137			58-62	132.8
		65-66	139			63 & Ov.	136.0
All Ages	128.91	All Ages	125	All Ages		All Ages	123.1
Tot. Entrants	19,339	Tot. Entrants	18,637	Tot. Entrants	62,000	Tot. Entrants	9,996

The averages for all ages signify very little for they depend upon the number of entrants at the different ages. Thus in Fisher's table only 13% of the entrants are younger than 36, so

that his general average is very high. In Mackenzie's and Goepp's tables, the largest number of entrants was about age 30 so that their general averages are quite low. For the individual age-periods, Fisher's and Mackenzie's averages are quite close to those set forth in Table 1, but Goepp's are a little lower. He noticed this and commented on it (10).

"The thought suggests itself whether the general reduction in the scale of living brought about by war conditions, particularly in the matter of eating and the use of alcohol, may not have had its influences on the averages obtained in the series."

On this hint those risks who were examined at the Home Office in New York City were separated into the years of issue and the results are set forth in Table 6.

TABLE 6

Years of Examination	Entrants	Average Systolic Pressure
1913	827	127.5
1914	973	126.0
1915	832	127.6
1916	829	125.4
1917	934	122.8
1918	883	123.7
1919	1076	123.9
1920	1203	125.4
1921	1338	126.5
1922 to June	580	125.2

The systolic blood pressure in New York City dropped decidedly in 1917 when we entered the war and stayed low during 1918 and 1919. It began to rise in 1920 and came back to the pre-war level in 1921. In the first five months of this year it seems to have fallen off again a little. In this table the averages for all ages combined are valuable for comparison, for the age-incidence is nearly the same for each year of issue.

The height seems to have almost no influence on systolic blood-pressure. Mackenzie (8) shows this for all ages com-

bined. The table below shows his results together with ours in two age-periods, 25-29 & 35-39.

TABLE 7
SYSTOLIC PRESSURE BY HEIGHT

Height	Mackenzie	Mutual Life	
		25-29	35-39
5 ft. 3 in. to 5 ft. 6 in.	125	124.2	125.5
5 " 7 " to 5 " 10 "	126	125.0	125.6
5 " 11 " & Over	126	125.3	126.7
Entrants	18,637	2,394	2,010

All of our cases were actually measured as they were examined at our Home Office. There is a rise of 1 mm. between the short and the tall in each age-period. By confining the comparisons in height to an individual age-period, the possibility of growth is avoided as a factor.

In submitting the mortality results of the cases with systolic pressure which have been analyzed in the preceding part, it must be recalled that the number of actual deaths has been much increased by the epidemic of influenza which struck this country in the fall of 1918 with one great recurrence in the spring of 1919 and another in the early part of 1920. Also this country's deaths from the casualties of war began to appear during 1917 and were very numerous during 1918. Our mortality tables, both the Medico-Actuarial and the American Men, are based upon years which did not include this epidemic. The Medico-Actuarial Table included the previous epidemic of 1889-1893 but this affected only a small part of the risks studied. Although the little war with Spain caused very high mortalities among our soldiers on account of abominable mismanagement the numbers involved were small and had but

TABLE 8

ISSUES OF 1907-1919 EXPOSED TO 1920—MORTALITY EXPERIENCE BY THE M. A. TABLE OF CASES WITH A RECORD OF SYSTOLIC PRESSURE

All Policy Years Combined

Systolic Pressure	15-29					30-44					45 & Over					All Ages				
	Deaths			Ratio	Infl. and War D.	Ratio Excl. Infl. and War D.	Deaths			Ratio	Infl. and War D.	Ratio Excl. Infl. and War D.	Deaths			Ratio	Infl. and War D.	Ratio Excl. Infl. and War D.		
	Act.		Expected.				Act.		Expected.				Act.		Expected.					
Below 101	9	5.63	150.8		4	88.0	4	4.93	81.2		81.2	81.2	31	3.39	57.2		13	11.64	93.3	64.6
101-110	94	9.00	139.5	37	62.8	91	103.0	91	103.0	87.6	18	78.3	505	895.87	62.7	27	216	248.98	92.8	57.1
111-120	664	711.95	93.2	230	59.7	701	1,032.08	701	1,032.08	76.6	189	58.3	200	274.87	72.8	4	1060	2,549.60	76.9	455
121-130	60	58.43	102.7	25	59.0	119	138.97	85.6	20	71.2	20	71.2	103	152.49	67.5	6	379	472.27	80.3	49
131-140	17	16.22	104.8	8	55.5	46	51.01	90.2	6	78.4	6	78.4	103	152.49	67.5	6	166	219.72	75.6	20
141-150	7	5.77	121.4	3	69.4	24	25.10	95.6	1	174.8	1	174.8	12	17.75	67.6	5	134	142.13	94.3	9
151-155	1	.37	274.0		274.0	4	2.29	174.8		45.9		45.9	3	7.03	42.7	2	17	20.40	83.3	87.9
156-160	1	.30			225.2	1	.64						3	7.87			3	7.87	83.1	12.7
Over 160	1	.44	225.2		225.2	1	2.18	45.9		62.2		62.2	10	14.15	113.1		18	16.77	107.3	107.3
Total	853	889.49	95.9	316	60.4	1080	1,361.05	79.4	234				973	1,441.01	67.5	46	2906	3,691.56	78.7	596
																				62.6

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little effect upon the general mortality of the country. The recent world war showed that hereafter the entire population must go to war, directly or indirectly. The deaths however will occur largely among the young men under 30.

We are assured by historians that there will be other wars, and by our own profession that there will be other epidemics of influenza. It is a doubtful expedient therefore to exclude deaths due to influenza and war. An actuarial friend says that this would be like a racing yacht built only for fair weather and smooth seas. As the Medico-Actuarial Mortality Table, which has been used for the calculation of the expected deaths, was built only for fair weather, it has seemed advisable to give the results both with and without the deaths due to influenza and the war. The deaths due to the war were all traumatic except a few which occurred in the prison-camps.

The results are set forth in Table 8. It will be noted that the actual deaths numbered 2,906 and the expected deaths 3,691.56. This gives a mortality ratio of 78.7%. The deaths from influenza and the war numbered 596, of which the large majority were due to influenza. When these are excluded, the mortality ratio drops to 62.6%. The cases have been divided into three sets of ages at entry, 15-29, 30-44 and 45 and over. The proportion of influenza and war cases differs markedly in these age-groups. In the youngest age-set they amount to 37%, in the middle to 22% and among those of 45 and upwards only to 5%.

As I showed two years ago in my paper on the "Value of the Medical Examiner's Opinion," (15), the influence of reliable medical examinations is conspicuous for five policy years and probably has a demonstrable effect even up to the tenth policy year. Now over 95% of the cases used in this study of systolic pressure were examined by very high-grade medical examiners, our Home Office staff and our medical referees with their immediate assistants. It seems advisable therefore to show the influence of systolic pressure on these cases after they have been insured for five years. Even though the number of deaths is very small in some classes, the general trend shows well. The following table sets forth the facts.

TABLE 9
Policy Years Six and Over

Systolic Pressure	15-29					30-44					45 & Over					All Ages				
	Deaths		Ratio	Infl. and War D.	Ratio Excl. Infl. and War D.	Deaths		Ratio	Infl. and War D.	Ratio Excl. Infl. and War D.	Deaths		Ratio	Infl. and War D.	Ratio Excl. Infl. and War D.	Deaths		Ratio	Infl. and War D.	Ratio Excl. Infl. and War D.
	Act.	Expect.				Act.	Expect.				Act.	Expect.				Act.	Expect.			
Below 101	1	.89	112.1	1		2	1.03	103.8		103.8	8	13.32	60.1		60.1	3	3.15	95.3	1	63.6
101-110	14	16.12	86.8	7	43.4	21	20.29	103.5	4	83.8	134	189.73	70.6	12	64.3	43	49.74	86.5	11	64.3
111-125	139	118.46	117.3	51	74.3	171	197.18	86.7	34	69.5	61	64.33	94.9	1	93.3	444	595.37	87.9	97	68.7
126-140	9	12.67	71.0	3	47.4	35	32.48	107.7	5	92.4	37	36.13	102.5	2	96.9	105	109.47	95.9	9	87.7
141-155	7	3.71	188.9	4	80.9	13	12.82	101.4	4	76.2	33	24.38	135.4	2	127.2	57	52.66	108.3	10	89.3
156-170		1.09				8	6.11	130.8		130.8	4	3.63	110.2	1	110.2	41	31.58	129.8	2	123.5
171-185		.08				1	.74	135.7		135.7	1	2.19	45.0	1	45.0	5	4.45	112.3	1	112.3
186-200		.18					.22	185.2			7	3.66	194.5			1	2.50	120.1		
Over 200		.14				1	.54									8	4.27	187.4		
Total	170	153.23	111.0	66	67.9	252	271.42	92.9	47	75.5	285	338.53	84.1	18	78.9	707	763.18	92.7	131	75.5

In order to get a more correct idea of the significance of these mortality ratios, they have been regraded on the basis of a par of 100% for the total of each age-group. For example the actual ratio of the age-group 15-19 for all policy years is 96%. This is called 100% and all the ratios above it for the different classes are increased proportionately. By this means the comparative relation of the mortality ratios to each other is correctly set forth, and it is much easier to distinguish the systolic pressures which show unusually high or low mortalities. Furthermore it will be of some value in assigning a numerical rating which properly belongs to each systolic pressure. Table 10 sets forth the facts both for all policy years and for policy years six and over.

In looking over Table 10 it will be noted that the class of 111-135 shows ratios below 100 throughout except in the age-period 15-29 among those insured for six policy years and over. Otherwise this class is very good.

TABLE 10

All Policy Years Combined

ASSUMED RATIOS BASED ON A TOTAL MORTALITY RATIO OF 100%

[illegible]

Policy Years Six and Over

Below 101	101.0		208.6	256.7			102.8	84.2
101-110	78.2	63.9	111.4	111.0	71.5	76.2	93.3	85.2
111-135	105.7	109.4	93.3	92.1	83.9	81.5	94.8	91.0
136-140	64.0	69.8	115.9	122.4	112.8	118.3	103.5	116.2
141-145	170.2	119.1	109.1	93.0	121.9	122.8	116.8	118.3
146-150			140.8	173.2	161.0	161.2	140.0	163.6
151-155			146.1	179.7	131.0	139.7	121.1	148.7
156-160					54.2		43.3	
Over 160			199.4		231.3		202.2	
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

In the age-period 15-29, class 101-110 is higher than 100 though it becomes much lower after the fifth policy year. In the class of lowest systolic pressure, those of 101 mm. and less, the number of deaths is too small but the tendency is distinctly towards larger mortalities. In the class 136-140 the ratio is above 100 but this seems due to deaths from influenza and the war, and it is good after the fifth policy year. The class 141-145 shows similar results but is bad after the fifth policy year. The number of deaths though is very small.

Age-period 30-44 shows very similar phenomena but class 141-145 gives poorer mortalities.

Age-period, 45 and over, shows that the classes of low pressure are distinctly better than the average, better even than the class 111-135. Class 141-145 is good except after the fifth policy year.

The idea that a systolic pressure above 140 is in the danger zone is not definitely established for the class 141-145, though the indications after the fifth policy year point that way. The class 146-150 shows results which call for distinct caution in acceptance. This caution has been manifested in the classes which show higher pressures. The highest group contains two sets of entrants, a few who showed a pressure slightly above 160 mm. but who were very good otherwise, and a larger number who showed a pressure above 160 mm. at first but which came down at subsequent readings below 160 mm.

TABLE II
SYSTOLIC PRESSURE—WOMEN ONLY

Ages	Bld. 8		Bld. 7		Bld. 6		Bld. 5		Bld. 4		Bld. 3		Total	
	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.
15-19	113.8	54	118.1	256	120.2	198	121.2	93	126.4	24	120.8	5	109.0	1
20-24	117.0	121	118.9	721	120.9	851	122.5	449	123.7	163	121.8	42	127.9	11
25-29	116.3	149	119.6	664	120.3	870	122.2	579	124.1	283	124.5	119	122.7	32
30-34	117.8	84	120.3	467	120.5	575	122.8	488	124.1	246	124.6	139	124.9	32
35-39	119.3	49	121.4	259	122.4	414	123.1	410	125.2	304	126.3	99	124.9	38
40-44	119.6	22	123.9	163	124.3	273	126.2	312	127.0	203	131.2	119	131.2	44
45-49	116.0	12	124.9	107	125.8	180	128.6	246	130.4	203	131.2	88	131.9	33
50-54	133.1	7	127.6	42	128.2	78	130.4	107	134.3	84	132.5	36	134.2	22
55-59	125.0	1	127.7	15	132.9	18	136.1	51	135.1	49	136.4	25	137.6	7
60 & Ov.			132.2	2	136.8	9	130.8	6	139.6	10	135.8	4	132.0	2
All Ages	117.1	499	120.2	2696	121.6	3466	124.0	2741	126.5	1585	127.2	676	129.4	222
													122.8	11,937

There were only 288 entrants in this class, of whom 185 were in the oldest age-period, and 172 in the heavier weight groups. The lighter weight groups showed in this class of 160 mm. and over a mortality ratio less than one-half that of the heavier weights but there were only 4 deaths in the higher weights and 14 in the heavier weights. The rather large mortality of this class of 160 mm. and over seems to be due quite as much to the over weight as to the high systolic pressure. Certainly the combination is bad.

SYSTOLIC PRESSURE—WOMEN

This study is based upon the women whose blood-pressures were recorded in the issues of 1907-1919 both inclusive. The remarks in the opening paragraphs of men's systolic pressure on page 1 will apply equally here. The analysis of the women by age and build-groups is set forth in Table II, which should be studied in connection with Table I.

The total entrants number 11,937 but Build-groups 9, 4 & 5 have been omitted, as they contained only 52 entrants though these entrants have been included in the totals. Women weigh less than men and more than 55% of them are in the lighter weight Build-groups 6, 7, 8 & 9 while only 40% of the men are in these groups. It will be noticed that the average systolic pressure is 1 or 2 mm. less than that for men up to age 40. After 40 the systolic pressure of women is quite equal to that of men, and it even may be 1 or 2 mm. higher.

If we eliminate from this table all the cases whose systolic pressure is above 140—as in Table 4 for men—we get the following results:

The first column under each build-group shows the average of those cases whose blood-pressure was 140 mm. or less, and the second column shows the percentage of those who were eliminated as their pressure was above 140 mm. The total eliminated number only 422, 3.5%, and the average pressure for all is lowered only .9 mm. As in the case of men, the older ages and the heavier build-groups show more marked reduc-

TABLE 12

SYSTOLIC PRESSURE—WOMEN—(140 MM. AND BELOW)

Ages	Bld. 8		Bld. 7		Bld. 6		Bld. 5		Bld. 4		Bld. 3		Total	
	Aver. Pres- sure	% Elim.	Aver. Pres- sure	% Elim.	Aver. Pres- sure	% Elim.	Aver. Pres- sure	% Elim.	Aver. Pres- sure	% Elim.	Aver. Pres- sure	% Elim.	Aver. Pres- sure	% Elim.
15-19	113.8		118.0	.4	120.2		121.0	1.1	126.4		120.8		119.1	.3
20-24	117.0		118.9		120.9	.2	122.2	.4	123.4	1.2	121.8		120.5	.3
25-29	116.3		119.5	.5	120.3		122.0	.5	123.8	1.1	124.2	1.7	120.7	.5
30-34	117.4	1.2	120.3		120.3	.9	122.3	2.0	123.5	2.4	124.2	2.2	123.6	6.3
35-39	119.3		121.2	1.2	121.9	2.2	122.3	3.4	124.7	2.6	124.5	8.1	128.0	15.8
40-44	119.6		122.7	4.3	122.8	7.0	125.2	5.4	125.2	8.7	127.1	10.1	126.9	20.5
45-49	116.0		122.4	10.3	124.1	7.8	126.3	11.4	126.7	18.7	127.1	21.6	128.1	18.2
50-54	127.0	28.6	125.6	7.1	124.7	16.7	127.6	15.9	130.3	23.8	128.3	22.2	130.3	22.7
55-59			126.6	6.7	128.6	22.2	130.9	31.4	128.3	38.8	132.0	28.0	129.5	30.7
60 & Ov.	125.0		132.2		129.7	33.3	128.2	16.7	135.8	20.0	131.7	25.0	131.6	20.6
All Ages	117.0	.6	119.9	1.1	121.1	2.0	123.2	4.0	124.9	7.4	125.3	8.9	126.1	15.3
Ent.	496		2,667		3,397		2,632		1,468		616		188	
													11,515	

tions of 2 to 5 mm. yet these changes are due almost entirely to risks whose pressure is between 141 and 150 mm., both inclusive, for there are only 38 women whose pressure is over 150 mm. and only 22 of these show a pressure above 155 mm. As in the case of men these changes depend upon the increasing proportion of risks with a pressure of 141 to 150 mm., in the older ages and the heavier build-groups and not upon the inclusion therein of risks with an unusually high pressure.

DIASTOLIC PRESSURE—MEN

This study is based upon the reports made by our examiners in the years 1916 to 1919. Ninety-five per cent of these reports on diastolic pressure were furnished by our examiners in New York City and our medical referees and their immediate assistants. All of these were trained to take the diastolic pressure at the very end of the 4th phase, practically just before the beginning of silence. Whether this really shows the pressure in the brachial artery after the pulse-wave has passed is open to discussion. It calls for the simplest technique and is determined more easily and regularly than any other point suggested for the diastolic pressure. It is certainly more easily determined than the end of the third phase as the sounds pass into the fourth phase. In many cases our examiners at the Home Office find it difficult to determine this change of sounds, and in some cases the fourth phase is entirely absent, but no one finds it hard to detect the last sound. In some abnormal conditions, especially aortic leakage, the sounds continue to be very low readings but these cases are unhealthy, have not been accepted as standard risks and are therefore not included in this study. The amount of pressure over which the fourth phase extends is about 2 to 6 mm. usually. As shown by Mackenzie (14) this range holds true for 75% of the cases. In 20% more it is from 7 to 10 mm. If larger, it is doubtful whether the examiner read the fourth point accurately. A wide cuff was used in all cases, and most of the

TABLE 13
DIASTOLIC PRESSURE—MEN ONLY

	Bld. 9		Bld. 8		Bld. 7		Bld. 6		Bld. 5		Bld. 4		Bld. 3		Bld. 2		Bld. 1		Bld. 0		Total	
	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.
70.8	3	755	102	76.9	1017	77.9	1,392	78.8	515	80.0	73	81.9	32	76.0	4	82.7	1	82.6	1	77.7	3,139	
75-19																					77.7	3,139
20-24																					79.6	7,321
25-29																					80.5	10,579
30-34	82.9	1	79.6	56	79.1	1337	79.7	3,853	80.9	3,307	81.8	1,321	82.9	544	82.8	138	85.4	21	73.8	1	81.5	11,709
35-39			80.0	45	79.8	1107	80.6	3,430	81.4	3,749	82.5	2,102	83.6	902	85.2	231	84.4	51			82.7	12,602
40-44			81.0	24	80.9	742	81.4	2,486	82.4	3,134	83.7	2,736	84.6	1,086	86.3	208	87.3	56			83.8	13,533
45-49			80.9	23	81.5	457	82.6	1,022	83.6	1,665	85.9	1,369	86.6	735	87.3	145	87.4	46	96.5	3	84.9	5,583
50-54			78.7	21	82.5	278	83.6	1,022	84.1	1,665	87.5	375	87.9	450	88.3	66	88.5	23			86.3	2,057
55-59			83.1	9	83.3	125	85.1	515	85.0	430	87.7	723	87.9	450	88.3	66	88.5	23			87.0	1,448
60 & Ov.			96.3	1	83.9	39	85.2	144	86.3	240	86.8	193	89.4	111	88.2	24	89.5	2			86.6	754
All Ages	73.8	4	78.8	346	79.6	6386	80.7	17,905	82.2	18,555	83.9	10,602	85.2	5176	86.1	1470	86.2	283	88.2	6	82.1	60,733

readings were taken on the Tycos manometer. All of the risks were accepted at standard rates.

The diastolic pressure is affected by weight and age in a manner similar to the systolic pressure. Table 13 therefore shows the average diastolic pressure arranged similarly to Table 2.

This table should be read in connection with Table I. If a man is 6 ft. tall and weighs 170 pounds we expect him to show a diastolic pressure of 80 mm. at age 22; of 82.4 mm. at age 37; and of 86.3 mm. after he is 60 years old. Similarly if a man is 32 years old and 6 ft. tall we expect him to show a diastolic pressure of 79.8 mm. if he weighs 140 pounds; of 81.4 mm. if he weighs 180 pounds; and of 85.2 mm. if he weighs 230 pounds.

Table 13 is based upon the records of 60,733 cases—all men—examined in the years 1916 to 1919 by first-class, well-trained examiners. Looking across the table it will be noticed that the diastolic pressure increases about 1 mm. for each build-group, until we reach build-group 3 after which there is increasing care in selection or perhaps a slight diminution in the functional activity of the circulation. Looking down a build-group it increases about 7 or 8 mm. from the youngest to the oldest. In proportion to its size the range of diastolic pressure, both in build-groups and in age-periods is nearly as large as in the systolic pressure. In the diastolic pressure there is no indication of the distinct rise which begins at age 40 in the systolic pressure. On the other hand the total increase in diastolic pressure is just as great proportionately as the total increase in systolic pressure. For the total diastolic increase is about 8 mm. and this is one-tenth of the diastolic average, while the total systolic increase is about 12 or 13 mm. and this also is about one-tenth of the systolic average.

Table 14 (below) is submitted as representing the diastolic pressure of healthy men "on the average," arranged according to age and build-group. It should be read in connection with Table I. It corrects the inequalities of Table 13 and expands it to cover all the build-groups at all ages.

TABLE 14

THEORETICAL DIASTOLIC PRESSURE FOR MEN

Build Groups

Ages	(9)	(8)	(7)	(6)	(5)	(4)	(3)	(2)	(1)	(0)	All Blds.
15-19	75	76	77	78	79	80	81	82	83	84	79.5
20-24	76	77	78	79	80	81	82	83	84	85	80.5
25-29	77	78	79	80	81	82	83	84	85	86	81.5
30-34	78	79	80	81	82	83	84	85	86	87	82.3
35-39	79	80	81	82	83	84	85	86	87	88	83.3
40-44	79	80	81	82	83	84	85	86	87	88	84.0
45-49	80	81	82	83	84	85	86	87	88	89	84.7
50-54	81	82	83	84	85	86	87	88	89	90	85.9
55-59	82	83	84	85	86	87	88	89	90	91	86.8
60 & Ov.	82	83	84	85	86	87	88	89	90	91	86.8
All Ages	78.9	79.9	80.9	82.0	83.0	84.4	85.4	86.0	87.0	87.8	83.5

If we eliminate from table 13 all those whose diastolic pressure is 95 mm. and higher, the results are similar to those in the systolic pressure. Build-group 9 is not shown for no cases were eliminated.

Only 4,186 cases—6.9%—are eliminated and the general average pressure is only lowered 1.3 mm. The large averages in the older ages and in the heavier build-groups are much reduced, so that in build-group 0 only 5 mm. and in 1 only 4 mm. separate the youngest from the oldest. Only 1,308 showed a diastolic of 100 mm. and upwards and most of these were just 100 mm. In fact only 175 showed 105 mm. and upwards. Nearly all of the cases eliminated therefore had a diastolic pressure of 95-100 mm.

DIASTOLIC PRESSURE—WOMEN

This study is based upon the women entrants who gave a record of the diastolic pressure in the issues of 1916 to 1919. The introductory paragraph in the study of Men's diastolic

TABLE 15

Age Periods	Bld. 8		Bld. 7		Bld. 6		Bld. 5		Bld. 4		Bld. 3		Bld. 2		Bld. 1		Bld. 0		Total	
	Aver. Pres-sure	% Elim-inated	Aver. Pres-sure	% Elim-inated	Aver. Pres-sure	% Elim-inated	Aver. Pres-sure	% Elim-inated	Aver. Pres-sure	% Elim-inated	Aver. Pres-sure	% Elim-inated	Aver. Pres-sure	% Elim-inated	Aver. Pres-sure	% Elim-inated	Aver. Pres-sure	% Elim-inated	Aver. Pres-sure	% Elim-inated
15-19	75.5	76.7	1.0	77.7	.9	78.3	2.7	80.0	3.6	80.3	12.5	76.0	2.8	84.6	82.7	77.5	1.3	82.7	77.5	
20-24	76.0	78.4	2.2	78.8	2.2	79.5	2.8	80.3	5.0	81.9	5.0	81.9	2.8	84.6	84.6	76.1	2.5	84.6	76.1	
25-29	76.6	79.0	1.7	79.2	2.6	80.2	3.9	80.9	5.4	81.9	6.4	81.4	8.7	84.9	84.9	76.9	3.5	84.9	76.9	
30-34	76.7	80.4	3.5	80.9	3.1	81.7	6.0	82.4	6.3	82.9	10.8	83.4	16.3	84.9	84.9	77.5	6.8	84.9	77.5	
35-39	77.0	80.8	3.0	81.4	6.0	82.1	9.0	82.0	11.1	83.7	13.0	83.0	15.8	84.8	84.8	78.1	9.2	84.8	78.1	
40-44	77.8	81.3	7.2	81.9	10.3	82.2	11.7	83.5	15.6	84.1	18.0	84.5	20.5	85.3	85.3	78.8	13.4	85.3	78.8	
45-49	81.4	81.8	9.6	83.1	12.8	83.7	14.7	84.1	17.6	84.6	23.3	85.3	23.4	84.4	84.4	83.8	16.7	84.4	83.8	
50-54	87.3	82.0	12.0	82.9	15.8	83.8	18.8	84.3	22.1	85.3	26.6	84.7	27.3	83.2	83.2	83.9	20.5	83.2	83.9	
55-59																				
60 & Ov.																				
All Ages	78.3	79.1	2.8	79.9	4.2	81.1	6.5	82.2	10.0	83.1	13.4	83.6	16.6	83.9	83.9	80.8	6.9	83.9	80.8	
Entrants	337	6,206		17,151		17,357	9,542	4,483	1,226	237	4	56,547								

TABLE 16
DIASTOLIC PRESSURE—WOMEN ONLY

Ages	Bld. 8		Bld. 7		Bld. 6		Bld. 5		Bld. 4		Bld. 3		Total	
	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.
15-19	76.4	24	77.1	121	77.2	109	78.3	8	82.7	2	82.7	1	77.2	311
20-24	76.7	49	77.3	333	78.3	373	80.0	86	80.5	22	82.6	3	78.2	1089
25-29	77.4	63	77.6	312	78.4	384	79.9	128	80.7	57	82.7	17	78.8	1219
30-34	77.1	34	79.0	188	79.6	227	81.0	122	82.7	73	81.8	15	80.3	894
35-39	78.8	14	81.0	105	80.6	195	82.4	133	85.5	36	84.0	14	81.8	676
40-44	77.7	12	83.0	73	82.1	116	83.6	89	85.4	44	88.2	13	83.4	487
45-49	77.7	7	83.4	46	84.0	74	86.3	87	86.9	38	88.5	13	85.0	369
50-54	88.6	3	81.9	13	86.3	36	89.5	42	85.4	12	88.7	9	87.1	153
55-59			80.8	3	87.4	10	91.0	20	85.3	11	92.0	3	87.8	68
60 & Ov.					87.9	3	89.5	3	90.5	1	90.5	1	89.8	10
All Ages	77.4	206	78.6	1194	79.6	1527	81.4	1225	83.7	296	85.0	89	80.5	5276

pressure applies equally here. The averages as affected by weight and age are set forth in Table 16.

The total entrants number 5,276 but build-groups 9, 4 and 5 have been omitted as they contained only 21 entrants, though these have been included in the totals. It will be noticed that the average diastolic pressure of women is about 1 mm. less than that of men up to age 40. Then for 10 years it is about the same, but at age 50 it increases quite rapidly, and afterwards it is substantially higher than that for men. The entrants after age 50 though are too few to assume that this increase over men's diastolic is confirmed. It is strengthened however by the rise in women's systolic pressure over men's systolic pressure at the same ages. Perhaps menstruation is responsible for the lower pressures, systolic and diastolic, among women younger than 40, but it does not seem reasonable to assign the menopause as the cause of the higher pressures after 50.

If we eliminate the cases whose diastolic pressure is 95 mm. and upwards, the results are shown in Table 17. The first column under each build-group shows the average pressure of those who still remain and the second column shows the percentage of those eliminated.

Only 215 cases—4.1%—have been eliminated and the general average has been reduced only .8 mm. But the high averages in the heavier weights and the older ages have been markedly reduced except in those of 60 years and over. Of the 215 eliminated, only 65 cases showed a pressure of 100 mm. and upwards.

For those who are statistically inclined the following Table 18 is submitted. It shows the medians and the standard deviations with the probable errors for the last columns in Tables 2, 11, 13 and 16. These are the basic tables of the systolic and diastolic pressures.

A careful study of Table 18 and the tables on which it is based shows that the frequency curves are nearly symmetrical though with a slight skewness toward the high pressures.

TABLE 17
DIASTOLIC PRESSURE—WOMEN—(94 MM. AND BELOW)

Ages	Bld. 8		Bld. 7		Bld. 6		Bld. 5		Bld. 4		Bld. 3		Total	
	Aver. Pres- sure	% Elim.	Aver. Pres- sure	% Elim.	Aver. Pres- sure	% Elim.	Aver. Pres- sure	% Elim.	Aver. Pres- sure	% Elim.	Aver. Pres- sure	% Elim.	Aver. Pres- sure	% Elim.
15-19	76.4		77.1	.9	76.9	1.8	77.5	2.3	78.3		82.7		77.0	1.0
20-24	76.7		77.1		78.2	.5	79.0	.4	79.3		82.6		78.2	.9
25-29	76.8	3.2	77.4	1.3	78.3	.8	79.6	1.9	79.8	.8	82.7		78.6	1.3
30-34	77.1		78.5	2.7	79.2	2.2	80.5	2.2	80.6	4.9	80.4	6.7	79.7	2.9
35-39	78.8		80.9	1.0	80.3	1.5	81.3	6.3	81.9	1.5	83.0	7.1	81.1	3.7
40-44	77.7		81.3	9.6	81.0	6.9	82.5	7.5	83.5	7.9	84.5	23.1	82.2	7.8
45-49	77.7		82.5	6.5	82.3	10.8	82.9	13.9	84.3	12.6	84.8	15.4	83.1	11.9
50-54	67.6	66.7	80.7	7.7	82.5	25.0	85.3	13.2	86.9	23.8	85.4	18.2	84.4	20.3
55-59			80.8		82.3	30.0	84.3	19.0	84.0	50.0	87.3	33.3	83.4	29.4
60 & Ov.					87.9		82.7	50.0	86.6	33.3	90.5		87.6	20.0
All Ages	77.0	1.9	78.2	2.0	79.0	2.8	80.6	4.7	81.6	7.1	82.4	8.1	79.7	4.1
Entr.	202		1,170		1,484		1,168		667		272		77	5,061

TABLE 18

Age Periods	TABLE 2		TABLE II		TABLE I3		TABLE 16	
	Median	Standard Deviation	Median	Standard Deviation	Median	Standard Deviation	Median	Standard Deviation
15-19	120.8 ± .07	7.18 ± .04	118.8 ± .24	7.11 ± .13	78.8 ± .10	6.81 ± .06	77.8 ± .28	5.84 ± .16
20-24	122.5 ± .04	6.85 ± .02	120.0 ± .14	7.98 ± .08	81.3 ± .07	6.85 ± .04	79.3 ± .16	6.34 ± .09
25-29	122.9 ± .04	7.21 ± .02	120.3 ± .13	8.14 ± .07	82.2 ± .06	7.14 ± .03	80.6 ± .16	6.56 ± .09
30-34	123.2 ± .04	7.34 ± .02	121.4 ± .14	7.63 ± .08	83.0 ± .05	6.94 ± .03	81.8 ± .18	6.54 ± .10
35-39	123.9 ± .04	7.92 ± .02	122.6 ± .19	8.76 ± .11	83.9 ± .06	7.19 ± .03	83.2 ± .22	6.83 ± .13
40-44	125.0 ± .05	8.62 ± .03	124.6 ± .23	9.49 ± .13	84.7 ± .07	7.47 ± .05	84.3 ± .27	7.05 ± .15
45-49	126.9 ± .07	9.62 ± .04	126.9 ± .30	10.35 ± .17	85.9 ± .09	7.82 ± .05	85.8 ± .32	7.20 ± .18
50-54	129.6 ± .10	9.87 ± .05	129.6 ± .44	10.18 ± .25	87.1 ± .12	7.82 ± .07	88.5 ± .53	7.80 ± .30
55-59	133.0 ± .14	9.74 ± .08	133.5 ± .68	10.35 ± .38	87.9 ± .18	8.08 ± .10	89.5 ± .86	8.38 ± .48
60 & Ov.	136.5 ± .20	10.40 ± .11	136.3 ± 1.50	10.34 ± .85	86.4 ± .26	8.61 ± .15	91.5 ± 1.67	6.25 ± .94
All Ages	124.1 ± .02	8.39 ± .01	121.9 ± .06	8.26 ± .04	83.4 ± .03	7.58 ± .01	81.8 ± .08	7.14 ± .05

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The material is compact, snugly grouped around the average and the deviations therefrom are small.

PULSE PRESSURE—MEN

This study is based upon examinations made at our Home Office from 1917 to August, 1922, and includes only risks accepted at standard rates. All readings were taken by auscultation, in a sitting posture with a mercurial manometer at the level of the heart. The diastolic pressure was recorded at the end of the 4th phase, the last distinct sound.

Table 19 shows the cases arranged according to age and build-groups.

This table should be read in connection with Table 1. If a man is 5 ft. 4 in. tall and weighs 140 pounds we expect him to show a pulse pressure of 43.8 mm. when he is 23 years old; of 42 mm. when he is 34 years old; and of 46.8 mm. when he is 57 years old. Similarly if a man is 41 years old and 5 ft. 4 in. tall, we expect him to show a pulse pressure of 41.4 mm. if he weighs 110 pounds; of 42.6 mm. if he weighs 138 pounds; and of 42.1 mm. if he weighs 180 pounds.

In studying the pulse pressure, we must remember always that it is not a real measure like the systolic and diastolic pressures, but only the difference between these two. All of its attributes depend upon these two real measures.

Thus the slight decrease from the youngest age-period up to 40 is due to the fact that the diastolic pressure increases about 3 mm. during that time while the systolic only increases about 2 mm.

The averages remain about 42 mm. until age 50, after which they increase markedly. The pulse pressure rises also slightly with increasing weight. Both these phenomena are on account of the faster rise in systolic pressure than in diastolic under these conditions.

Although the average pulse pressure shows very small changes in the different age-periods the cases upon which this study is based show that the range between the extremes of

TABLE 19
(FROM 1917 TO AUGUST 1922)—HOME OFFICE CASES—PULSE PRESSURE—(MEN ONLY)

Ages	Bld. 8		Bld. 7		Bld. 6		Bld. 5		Bld. 4		Bld. 3		Bld. 2		Bld. 1		Bld. 0		Bld. 6		Bld. 7		Bld. 8		Total	
	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.	Aver.	Entr.
15-19	40.5	11	41.9	69	42.4	97	45.0	43	46.3	6	48.5	4	44.0	1	49.0	2	42.9	233								
20-24	38.5	8	41.9	141	43.2	293	43.8	181	41.8	45	43.3	17	43.1	9	45.3	3	43.0	697								
25-29	41.3	9	42.7	190	42.9	379	42.8	276	44.3	127	42.6	50	42.8	35	43.8	6	43.0	1072								
30-34	41.5	10	40.3	168	41.7	342	42.0	331	42.2	102	41.3	97	44.0	43	42.7	59	41.8	1774								
35-39	41.5	7	41.3	111	42.2	260	42.2	280	42.6	205	43.0	121	41.4	39	40.7	22	42.2	1781								
40-44	41.5	7	41.3	111	42.2	260	42.2	280	42.6	205	43.0	121	41.4	39	40.7	22	42.2	1781								
45-49	41.5	7	41.3	111	42.2	260	42.2	280	42.6	205	43.0	121	41.4	39	40.7	22	42.2	1781								
50-54	41.5	7	41.3	111	42.2	260	42.2	280	42.6	205	43.0	121	41.4	39	40.7	22	42.2	1781								
55-59	41.5	7	41.3	111	42.2	260	42.2	280	42.6	205	43.0	121	41.4	39	40.7	22	42.2	1781								
60 & Ov.	44.0	1	46.9	7	50.3	16	51.7	24	46.1	18	47.0	8	56.7	3	50.7	3	49.2	78								
All Ages	41.0	56	41.6	836	42.6	1772	43.1	1633	43.2	931	43.6	502	43.4	251	42.9	86	42.8	6071								

high and low is much greater than in the systolic or diastolic pressure in proportion to its size. Thus the lowest pulse pressure was 20 and the highest 64. Between 20 and 64 is a range of 44 mm. which is actually larger than the average pulse pressure for all ages and also for each age-period up to 50 years. On the other hand the range of systolic pressures was from 95 mm. to 160 mm. This represents a gap of 65 mm. which is barely one-half of the average systolic pressure. The diastolic range was from 50 mm. to 103 mm.; a gap of 53 mm. which is only a little more than one-half of the average diastolic pressure.

Not only is the range of deviation from the average much greater in the pulse-pressure but also the amount of deviations. This is well set forth in Table 20 which shows the coefficient of variation for the three pressures. The coefficient of variation is obtained by dividing the standard deviation by the average, and is a very good measure of the extent and quantity of the deviations from the average. These figures are obtained from the material which is used in the study of the pulse pressure only.

TABLE 20
COEFFICIENTS OF VARIATION

Age Periods	Systolic	Diastolic	Pulse
15-19	8.3 - .2	9.1 - .3	17.7 - .6
20-24	8.7 - .1	10.4 - .2	17.5 - .3
25-29	8.6 - .1	9.9 - .2	17.1 - .3
30-34	8.8 - .1	9.7 - .1	17.9 - .3
35-39	8.9 - .1	9.5 - .1	18.1 - .3
40-44	9.0 - .1	9.7 - .2	17.3 - .3
45-49	9.3 - .1	9.9 - .2	17.7 - .4
50-54	8.9 - .2	8.9 - .3	16.8 - .5
55-59	8.4 - .2	8.1 - .4	16.4 - .7
60 & Ov.	8.6 - .3	8.8 - .5	13.8 - .8
All Ages	9.0 - .04	10.0 - .1	17.7 - .1

The diastolic coefficients are slightly larger than the systolic but the coefficients of the pulse pressure are much larger than either of the others. They are more than twice as large as the systolic in some age-periods, and nearly twice as large as the diastolic coefficients. This is not unnatural since the pulse pressure is the resultant of the two variables, the systolic and the diastolic.

We are all impressed by the fact that the average pulse pressure remains nearly at a level up to age 50 and after that only rises a few millimeters while both the systolic and diastolic increase steadily with age and the increase in millimeters is larger than in the pulse pressure. At first this seems to indicate that the pulse pressure is the more stable and less liable to erratic fluctuations. But even this stability is fictitious if we take into account the average number of millimeters in each of the pressures.

This marked variability of the pulse pressure with a range from 20 mm. to 60 mm. in apparent health casts some doubt upon its clinical significance and its value for our insurance purposes. A favorite ratio has been pulse pressure 1: diastolic pressure 2: systolic pressure 3. This is the so-called 1: 2: 3: ratio and is probably derived from the averages. The pulse pressure of 42 which is obtained from a diastolic of 84 and a systolic of 126 is ideal. But is there any reason why a risk should be called unhealthy when the diastolic is 84 and the systolic 104, or when the diastolic is 90 and the systolic 110? Yet in these cases the ratios would be 1: 4: 5 and 1: 4.5: 5.5. Should a risk be considered unhealthy when the systolic is 140 and the diastolic 70? This gives a pulse pressure of 70 and the ratio becomes 1: 1: 2. Doubtless these unusual pulse pressures call for very careful examinations of the cardiovascular apparatus. In the last case the medical examiner should look most carefully for any evidence of aortic leakage, and in both sets of cases for any signs of arteriosclerosis. If he certifies that there is no evidence of any impairment in the heart or vessel after careful examination, these unusual cases should not be regarded as unhealthy. Perhaps it

may be said that pulse pressures less than 30 mm. or larger than 56 mm. should be regarded as a warning signal to examine the heart and vessels with great care, but otherwise its value to us for life insurance seems quite limited. Much research will have to be made and many facts accumulated before it can be determined whether it has any real significance.

If cases with a pulse pressure larger than 54 are eliminated, the results are set forth in Table 21, which shows in the first column under each build-group the average pressure of those cases who were left in the group, and in the second column the percentage of those cases who were eliminated.

Only 382 cases—6.3%—are eliminated and the average, pulse pressure is reduced only 1 mm. yet the very high averages in the older ages and the heavier weights have been greatly reduced. This probably means that the cases eliminated are those with larger systolic pressure which occurs naturally under these conditions. I doubt if it has any other significance but even this should make us scan a pulse pressure of 55 mm. and upwards with care.

Pulse pressures of less than 30 mm. numbered 163, 2.7%. The percentage of these cases range from 1.7% at age 15-19 up to a maximum of 3.3% at age 30-34 and then down to 1.6% at age 55-59. There is no significance in these figures and only from our general knowledge can it be said that a pulse pressure less than 30 mm. should call for special information on the condition of the myocardium and the blood vessels.

An effort has been made to use the product of the pulse pressure and the pulse rate as an index of the work of the heart. Addis has written an excellent paper on this product (12). Before it can be used for insurance purposes, it will have to be studied carefully. He says:—

“The striking difference between the averages of normal individuals under basal and daytime conditions are the clearest illustration of the necessity for uniformity in the conditions under which the observations are made. It is not possible to use the basal normal for the evaluation of pressures obtained in patients in the morning if they have been out of bed even

TABLE 21
(FROM 1917 TO AUGUST, 1922)—HOME OFFICE CASES—PULSE PRESSURE—54 AND BELOW (MEN ONLY)

Ages	Bld. 8		Bld. 7		Bld. 6		Bld. 5		Bld. 4		Bld. 3		Bld. 2		Bld. 1		Bld. 0		Bld. 4		Total	
	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated	Aver. Pres- sure	% Elim- inated
15-19	39.0	9.1	41.0	4.3	41.0	4.1	42.7	16.3	44.4	16.7	48.5	5.9	43.1	44.0	43.1	42.5	42.5	42.5	42.5	42.5	41.9	6.9
20-24	38.5		41.2	4.3	42.2	6.8	42.6	8.3	41.4	2.2	42.5	5.9	43.1	43.1	43.1	42.5	42.5	42.5	42.5	42.5	42.0	6.2
25-29	41.3		42.3	2.1	42.0	6.1	42.0	4.7	43.2	7.9	42.3	2.0	42.4	42.3	42.4	42.3	42.3	42.3	42.3	42.2	4.9	
30-34	41.5		39.7	3.6	41.2	3.5	40.9	6.9	41.0	6.8	40.8	3.1	42.5	42.5	42.5	42.5	42.5	42.5	42.5	41.8	5.3	
35-39	41.3		40.3	5.4	41.4	4.8	41.1	6.6	42.0	3.9	42.1	5.8	40.9	40.9	40.9	40.9	40.9	40.9	40.9	40.9	41.4	5.2
40-44	42.6		40.2	6.8	41.1	5.2	41.0	8.3	42.1	5.5	42.0	12.8	42.0	42.0	42.0	42.0	42.0	42.0	42.0	41.5	6.1	
45-49	42.6		41.3	15.8	43.1	11.8	43.6	8.0	44.2	12.9	44.8	17.9	44.8	44.8	44.8	44.8	44.8	44.8	44.8	43.5	12.5	
50-54	45.0	33.3	41.9	11.1	43.6	17.4	44.5	18.6	46.3	14.3	44.8	33.3	44.8	44.8	44.8	44.8	44.8	44.8	44.8	43.5	12.5	
55-59	44.0		41.1	11.1	43.6	17.4	44.5	18.6	46.3	14.3	44.8	33.3	44.8	44.8	44.8	44.8	44.8	44.8	44.8	43.5	12.5	
60 & Ov.	44.0		46.9		47.2	31.3	48.3	33.3	44.0	16.7	45.4	12.5	45.4	45.4	45.4	45.4	45.4	45.4	45.4	46.5	24.4	
All Ages	40.4	3.6	41.0	4.1	41.7	5.5	42.0	7.4	42.3	6.3	42.4	8.0	42.1	42.1	42.1	42.1	42.1	42.1	42.1	41.8	6.3	
Entrants	54		802		1,674		1,512		872		462		230		81		5,689					

for a moment. The normal values for daytime measurements cannot be taken as a standard for observations made on patients who are standing or sitting, or on those who have just walked up a flight of stairs. The variability of normal blood-pressure under such conditions is not known."

All of his readings were taken lying down while practically all insurance records are taken sitting up. His coefficients of variation for systolic pressure were 11 for basal and 13 for daytime; for diastolic pressure 14 for both basal and daytime; and for pulse pressure 30 for basal and 28 for daytime. These coefficients are decidedly larger than those in Table 20 and tend to show that the blood-pressure in all aspects is more variable when lying than sitting. Perhaps some of his "normal" cases would be classified as people with idiopathic vascular hypertension, described by O'Hare (13). A careful reading of his and other papers has convinced me that the method of obtaining the blood-pressure as practiced by insurance companies is trustworthy and reliable for people in average good health. Doubtless food, exercise and excitement have some effect but they are rarely sufficient to raise the blood-pressure in a healthy man above the point of acceptance by a life insurance company. If an applicant's blood-pressure is too high at the first examination and remains so after he has been properly soothed and quieted, it does not often come down to acceptable figures at a second examination, unless treatment is taken. Very few readings are so low on account of dread of the examination as to need a second test, for life insurance has become a commonplace to most applicants.

SUMMARY

1—The average systolic blood-pressure of healthy men is set forth in Table 3, which is to be read in connection with Table 1. It increases decidedly both with age and with weight. Nearly one-half of the increase in both cases is due to the increasing proportion of systolic pressures between 141 and 150 mm. In connection with the practice of companies using a numerical rating, like the New York Life, which charges an excess mortal-

ity for a systolic pressure of 140 mm. in ages below 40, a question arises whether any systolic pressure above 140 mm. should not be suspected of pathological possibilities. Certainly we have good reasons to regard any pressure above 150 mm. as in the danger-zone for life insurance.

Pressures below 100 mm. are rare in life insurance. They will usually be found in the very young and thin, and life insurance has shown that this combination is prone to tuberculosis. To some extent this holds true also for pressures below 110. Among those who are not young these low pressures do not seem to be associated with increased mortality, though probably these risks have been very carefully selected. The matter is complicated by the fact that recent mortality reports includes the influenza epidemic, and this caused high mortalities in the ages below 30. The decision as to the low safe limit of systolic pressure must be regarded as still *sub judice*.

The systolic pressure of women is set forth in Table 11, which should be read in connection with Table 1. It is a little lower than men's up to age 40 partly for the reason that women weigh less up to this age. After that it is a little higher than men's and Table 12 shows that it behaves like men's with reference to pressures over 140 mm.

2—The diastolic pressure of healthy men is set forth in Table 14, which should be read in connection with Table 1. It increases with weight and age in about the same proportion as the systolic pressure. It is possible that a diastolic pressure above 94 mm. is in the danger-zone as Table 15, points in this direction.

The diastolic pressure of healthy women is set forth in Table 16. It is a trifle lower than men's up to age 40 and a trifle higher after age 50. Table 17 corroborates the significance of diastolic pressures above 94 mm.

3—The pulse pressure of healthy men is set forth in Table 19. As it is not a real measure but only the difference between the systolic and diastolic pressures, all of its attributes depend upon these. It is very variable and its value to life insurance seems to be only incidental and not substantial.

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Probably life insurance and general medicine will never regard blood-pressures in the same light. Life insurance sees only people who are healthy or at least think they are. Even the highest pressure of fat elderly people is below 140 mm. on the average, if they are acceptable for life insurance. This also means that practically as many are below 140 mm. as above and we have seen that of those above 140 mm. nearly all of them are below 150 mm. General medicine on the other hand sees those who feel that they are sick. If their illness is due to blood-pressure, it is usually high, frequently as high as 200 mm. or more. General medicine knows that these high pressures will come down to 170 mm. or 180 mm. by appropriate treatment and many of them live for years. There are examples among our own associates. But medicine does not realize that a small increase in the number of deaths per year means a great difference to life insurance. At age 50 we only expect 14 to die in the following year out of 1000 living and we call that 100% mortality. If 28 die, our mortality jumps up to 200%. At age 60, if the number of deaths among 1000 living increases from 26.69 to 40.04 the mortality increases to 150%. If a practitioner should see 1000 patients with high blood-pressure at age 60 and bet with himself that 974 would survive the year and only 960 did survive he would not feel downcast. In fact he would probably point to the record with pride and boast of his ability in prognosis. But life insurance would have to tell him that his mortality was 150% in that group and a medical director who never made a better guess than that would not keep his position for long. General medicine would look complacently at the living but life insurance would ruefully regard the dead for forty claims would have to be paid instead of the twenty-six expected.

REFERENCES

1. *Medico-Actuarial Mortality Investigation*, vol. i, pp. 120-121.
2. ROGERS AND HUNTER. *Proceedings of the Association of Life Insurance Medical Directors*, vol. viii, 1921-1922, p. 130.
3. WALTER C. ALVAREZ, 1920, *Archives of Internal Medicine*, xxvi, p. 381.

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4. BARACH & MARKS. *Archives of Internal Medicine*, 1914, xiii, p. 648.
5. B. SMITH. *J. A. M. A.*, 1918, lxxi, p. 171.
6. SORAPURE. *Lancet*, 1918, ii, p. 841.
7. J. W. FISHER. *J. A. M. A.*, 1914, lxxiii, p. 1754.
8. LEWIS F. MACKENZIE. *Proceedings of Association of Life Insurance Medical Directors*, 1915, p. 221.
9. ROGERS and HUNTER. *Proceedings of Association of Life Insurance Medical Directors*, 30th Annual Meeting, 1919, vol. vi, p. 92.
10. GOEPP. *Penn. Med. Journal*, 1919, xxii, p. 295.
11. JOHN W. FISHER. *Proceedings of Association of Life Insurance Medical Directors*, 31st Annual Meeting, vol. vii, 1920-1921, p. 21; also 26th Annual Meeting, 1915-1916, p. 203.
12. T. ADDIS. *Archives of Internal Medicine*, 1922, vol. i, p. 539 and vol. ii, p. 240.
13. JAMES P. O'HARE. *Am. Journal of Medical Sciences*, vol. clix, p. 369.
14. L. F. MACKENZIE. *Medical Record*, 1920, vol. 97, p. 1029.
15. BRANDRETH SYMONDS. *Proceeding of the Association of Life Insurance Medical Directors*, Vol. VII, p. 29.

REPORT OF BLOOD PRESSURE COMMITTEE

By J. W. FISHER, M.D.

Medical Director The Northwestern Mutual Life Insurance Company

Submitted herewith is the report of the mortality of 4165 applicants rejected for insurance by the Northwestern Mutual Life Insurance Company on account of a persistent systolic blood pressure, palpatory method, of 10 to 50 mm. or more over the average for the age, with no other impairment, during the years 1907-1920, carried to the Anniversary in 1921. Table I shows the mortality by ages and Table II by millimeters of pressure over the average for the age, mortality computed for the first year, the first five years, and for all years. In Table II, it will be seen that there were 525 cases with an average blood pressure of 12 mm. over the average for the age, with 26 deaths, where only 19 were expected—mortality 136.1%.

By referring to previous reports of this Committee, it will be seen that the mortality in this class is in excess of the 520 cases accepted during the years 1907-1910, ages 40 to 60, with an average blood pressure of 153 mm., computation to Anniversary in 1919, with a mortality of 114.4%. On 283 of this group, ages 45 to 53, the mortality was 136%, while this same class, as shown in our 1915 report, the mortality was 127%, ages 40 to 60, by the M. A. Table—bear in mind this group was approved during the time when few examiners were trained in the accurate use of the manometer and the majority of cases were accepted with only one reading, which, in many cases, no doubt, the hypertension was accidental and not an accurate reading. Furthermore, we find that 48 cases had gone off the books by lapse, surrender and maturity, which, in our mortality compilation, we treated as still living, no effort having been made to determine how many of these cases had died since their insurance had terminated, so that the results are not as accurate as that shown in the 525 cases rejected in Table II, with a persistent pressure of 10-14 mm., after repeated readings, showing a range of little or no fluctuation, so that the latter is much more reliable data.

Your attention is called to our report of October 7, 1915, pages 213 to 215, transactions of the Association for the years 1915 to 1916, also the conclusions reached with respect to hypertension. In addition to the 507 deaths shown in Tables I, II, and III, we learned that 839 of the 4165 cases under observation have developed impairments, about 85% of which are cardio-vascular-renal disease and continued hypertension—this, added to the 541 impairments previously reported, giving us knowledge of 1132 of these, though living, are impaired. We also have a record of 751 cases previously declined by the Company on account of high blood pressure only, where after varied subsequent treatment, correction in diet, change in mode of living, etc., and in some cases the hypertension was found to be temporary or accidental, the blood pressure eventually became normal, and they received insurance in this and other companies. 346 of this group were insured in this Com-

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pany. The mortality, thus far, is found to be favorable, and we assume that the remaining 405 similar cases, in which the blood pressure was "q'd" and received insurance in other companies, will also show like favorable results. In our investigation, there were 377 declined cases that could not be located. We assumed that the mortality on these would be similar to that shown in the Tables.

We have made no effort since 1915 to determine the mortality of risks declined in which there was a high blood pressure and one or more other impairments. We determined in 1915 that the mortality of this class was about 40% higher than the class in which there was a high blood pressure and no other ompairment.

For convenience, we have included in Table III, a summary of practically all of the data furnished by the Committee on the subject during the past fifteen years. The data shown in this Table is in graphic form, consisting of low blood pressure, diastolic blood pressure, the average blood pressure of 142 mm., ages 40 to 60, and average blood pressure of 153 mm., ages 40 to 60, details of which will be found in our last report to this Association, vol. vii, pages 17 to 22, the remaining data shown in Table III consisting of 4165 rejected cases in which high arterial tension was the only impairment, shown by ages and also by the number of millimeters over the average for the age. In this investigation, no effort was made to determine the status of the 1449 cases declined in the years 1919-1920, owing to the short time elapsed since rejection. We, however, learned of 11 deaths occurring in 1919 and 8 in the year 1920, which occurred prior to the Anniversary date in 1921—see Table IV, showing the ratio per cent of deaths for each of the fourteen years covered by this data. We have in addition, notice of 35 deaths which occurred since the Anniversary date in 1921, covered by the data shown in Table I.

TABLE I

ACTUAL MORTALITY RISKS REJECTED DUE TO HIGH SYSTOLIC BLOOD PRESSURE ONLY
(PALPATORY METHOD) DURING THE YEARS 1907 TO 1920, INCLUSIVE

Mortality Computed to Anniversary in 1921 by the American Men Table

Ages at Entry	Number of Lives	First Year		First Five Years		After Five Years		All Years		Ratio %
		Deaths		Deaths		Deaths		Deaths		
		Actual	Expected	Actual	Expected	Actual	Expected	Actual	Expected	
16-20	743	5	2.0	16	7.4	2	1.8	18	9.2	195.9
20-30	731	5	2.3	28	9.8	6	3.3	34	13.1	259.5
40-40	1196	20	6.0	106	32.1	75	22.0	181	54.1	334.6
50-60	1495	24	15.3	175	85.6	99	58.0	274	143.6	190.8
Total	4165	54	25.6	325	134.9	182	85.1	507	220.0	230.5
Ratio %		210.9		240.9		213.9		230.5		

TABLE II

ACTUAL MORTALITY RISKS REJECTED DUE TO HIGH SYSTOLIC BLOOD PRESSURE ONLY
(PALPATORY METHOD) DURING THE YEARS 1907 TO 1920, INCLUSIVE

*Mortality Computed to Anniversary in 1921 by the American Men Table, ages 16-60
inclusive*

Mm. over Average Blood Pressure for age 1907-1920	Number of Lives	First Year		First Five Years		After Five Years		All Years		Ratio %
		Deaths		Deaths		Deaths		Deaths		
		Actual	Expected	Actual	Expected	Actual	Expected	Actual	Expected	
		Actual	Expected	Actual	Expected	Actual	Expected	Actual	Expected	
+10-14	525	3	3.4	21	13.6	5	5.5	26	19.1	136.1
+15-24	1685	10	9.0	88	45.8	37	22.2	125	68.0	183.8
+25-34	909	7	5.8	66	32.0	45	22.3	111	54.3	204.4
+35-49	657	12	4.6	71	26.6	50	22.1	121	48.7	248.5
+50	389	16	2.8	79	16.9	45	13.0	124	29.9	414.7
Total	4165	54	25.6	325	134.9	182	85.1	507	220.0	230.5
Ratio %		210.9		240.9		213.9		230.5		

TABLE III

Summary of the Mortality Experience of The Northwestern Mutual Life Insurance Company, with respect to the Palpatory Systolic and Auscultatory Diastolic Blood Pressure.

Years of Entry	Range Arterial Tension	Age at Entry	Number of Lives		Actual Deaths	Ratio Actual to Expected Mortality (American Men Table)				
			Accepted	Rejected		100 %	200 %	300 %	400 %	%
1912-1920	Low Blood Pressure 100 mm. and under	16-60	3389		26					**35.0
1915-1920	Diastolic Blood Pressure 95mm. and above	16-60	1244		22					78.6
1907-1910 to anniversary in 1919	Blood Pressure Av. 140mm.	40-60	2610		310					94.0
	Blood Pressure Av. 150mm.	40-60	520		90					114.4
		16-29		743	18					195.7
		30-39		731	34					259.5
		40-49		1196	181					334.6
		50-60		1495	274					190.8
	Total	16-60		4165	507					230.5
Risks Rejected for High Blood Pressure Only Years 1907-1920—Mortality Computed to Anniversary 1921	Min. over average +10-14			525	26					186.1
	+15-24			1685	125					183.8
	+25-34			909	111					204.4
	+35-49			637	121					248.5
	+50			389	124					414.7
	Total	16-60		4165	507					230.5

*The Northwestern Mutual Life Insurance Company's General Mortality Experience about 60% of the Table

**Estimated Mortality

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TABLE IV

RISKS REJECTED HIGH PALPATORY SYSTOLIC BLOOD PRESSURE ONLY

Mortality to Anniversary in 1921

(A. M. TABLE)

Years of Entry	Number of Lives	DEATHS		Ratio %
		Actual	Expected	
1907	21	15	3.4	441.2
1908	52	32	9.4	340.4
1909	85	40	14.8	270.3
1910	108	48	18.9	254.0
1911	140	38	19.3	196.9
1912	320	72	34.7	207.5
1913	314	56	28.3	197.9
1914	239	47	17.8	264.0
1915	347	43	20.4	210.8
1916	373	43	18.0	238.9
1917	391	33	14.1	234.0
1918	326	21	8.4	250.0
1919	587	11	7.9	139.2
1920	862	8	4.6	173.9
TOTAL	4165	507	220.0	230.5

CAUSES OF DEATH OF THE 507 CASES AS SHOWN IN
TABLE I

CAUSES OF DEATH	Entry Ages 16-39 Number	Entry Ages 40-60 Number	Total
Anemia, Pernicious.....	0	3	3
Angina Pectoris.....	2	18	20
Apoplexy.....	5	104	109
Appendicitis.....	2	4	6
Arteriosclerosis.....	1	39	40
Bladder Operation.....	0	1	1
Cancer.....	3	17	20
Casualties.....	4	5	9
Diabetes.....	0	4	4

CAUSES OF DEATH OF THE 507 CASES AS SHOWN IN
TABLE I—Continued.

CAUSES OF DEATH	Entry Ages 16-39 Number	Entry Ages 40-60 Number	Total
Duodenal Ulcer.....	0	4	4
Gallstones.....	0	3	3
Heart Disease (Organic).....	5	83	88
Hernia Operation.....	0	2	2
Influenza-Pneumonia.....	6	1	7
Insanity.....	1	3	4
Kidney Operation.....	0	1	1
Liver Disease.....	0	3	3
Locomotor Ataxia.....	0	1	1
Lungs Edema.....	1	1	2
Meningitis, Cerebral.....	1	1	2
Nephritis.....	9	97	106
Paralysis.....	0	4	4
Pleurisy.....	0	1	1
Pneumonia.....	4	10	14
Prostatic Hypertrophy.....	0	1	1
Sudden Death.....	0	8	8
Suicide.....	2	5	7
Syphilis.....	0	2	2
Tuberculosis.....	2	3	5
Tumor, Brain.....	2	2	4
Miscellaneous.....	2	24	26
TOTAL.....	52	455	507

DR. WM. EVELYN PORTER.—When called upon by our President to open the discussion of Dr. Symonds' paper, I informed him that a careful review of the galley proof demonstrated the fact that the subjects involved had been so thoroughly covered and were so comprehensive as to leave little opportunity for discussion from the standpoint of our own Company. As members of this Association we are quite familiar with the admirable character of the Doctor's contributions and he has certainly maintained his accustomed standard in this paper.

Reviewing the article, it will be noted that in the earlier years many of the blood pressure readings were obtained by

palpation, whereas since 1915 nearly all have been taken by auscultation. It is the latter group that is of special interest and value for present consideration and future comparison.

As stated by Dr. Symonds, the material for this study consisted of records of risks accepted at standard rates by The Mutual Life Insurance Company. Over 95% of the readings were taken by our New York City staff under my supervision, and by our Medical Referees and their assistants, most of whom have received personal instruction from me, as to the details of procedure to be followed in our work. Whereas the Tycos manometer was employed in outside examinations and by most of the referees and their assistants, the baumanometer was used with many of the Home Office cases. With many of these latter cases the sphygmometroscope was used, adding definitely to the uniformity of results. Applicants were seated with the manometer about the level of the heart, a 5-inch band applied, with the arm slightly flexed and all essentials of correct technique were followed with precision and care. I can personally vouch, therefore, for the accuracy with which the work was done. The diastolic readings were taken at the vanishing point or just prior to the final disappearance of sound, which represents the very end of the fourth or essentially the fifth phase.

In instructing examiners in this respect, I have used the expression "just prior to the vanishing point" advisedly, in order to avoid undue effort on their part to trace the faintest resemblance of sound which might lead to an incorrect record.

Reference to the transactions of the twenty-eighth annual meeting of this Association, held in 1917, will show that I stated that during the previous year I had systematically taken both the palpatory and the auscultatory readings of the systolic and found variations of from 6 to 12 mms. I suggested the advisability of taking both readings, but with added experience and efficiency this procedure is of value only in occasional cases where the sounds are very indistinct. I further stated that for a period nearly two years prior to that time I gave instructions to my medical examiners and to the referees

to take their diastolic readings at the vanishing point, believing that we would obtain more uniform results than we could in trying to determine the change from the loud sharp to the dull muted sound which represents the fourth phase. The experience of the past five years has convinced me of the greater practical value of this procedure.

In studying the general subject of the auscultatory method I find that even among many expert physicians there is a very vague knowledge of what constitutes the five phases and in order to have a clear uniform understanding of what is represented by each phase, I have adopted the following classification:

FIRST PHASE

Clear-cut, shot-like, snapping sound.

SECOND PHASE

Loud rough stenotic sound.

THIRD PHASE

Partial or complete disappearance of sound with return similar to first phase, though not quite so loud.

FOURTH PHASE

Change of character of sound from a sharp pistol-shot to a muted or dull type.

FIFTH PHASE

Final disappearance of any sound.

The sounds heard during the first phase described as clear-cut, shot-like, and snapping, vary considerably in intensity, and should not be confused with a few vague sounds occasionally heard at the beginning of an examination at an absurdly high point, disappearing at once and followed by the sound which represents the real first phase. The constant finding on two or three trials of the sound described gives the true first phase indicating the systolic reading.

The change described as the second phase is often not clearly in evidence but the partial or complete disappearance of sound at the beginning of the third is usually reasonably well marked and is followed at once by a return of character similar to the first phase, though not quite so loud. With examiners other than experts, this change might be accepted as the fourth phase, the reading recorded leading to a higher and incorrect figure, where the fourth phase is accepted as the standard.

The fourth phase is the change of character of sound described by Theodore C. Janeway as "the point of sharp change from the loud pistol-shot sound to the dull sound," which he designated as the end of the third or beginning of the fourth phase.

The fifth phase represents the final disappearance of all sound.

Whereas in routine insurance or clinical work it will often be impossible to determine these various phases, it is well to have a clear knowledge of what each is supposed to constitute.

Owing to this very difficulty of determining these phases, particularly the fourth, some years ago I had a number of our Home Office examiners study carefully the presence or absence of the change described as the fourth. As a result, it was demonstrated beyond question of doubt that in a very large percentage the change was ill defined or not determined. The final disappearance of sound can be determined in nearly all cases and therefore affords a more constant point at which to record the diastolic reading. Occasionally there will be difficulty in detecting the disappearance, and absurdly low records will result; but with such findings, aortic leakage will often be detected, demanding careful re-examination of the heart. The percentage of difficulty at this phase, however, is much less than that due to the ill-defined character of the fourth. Nearly all of the medical schools teach the students to determine the fourth phase as the standard, and where the observations are accompanied by careful tracings with the electrocardiograph, the changes in both observations will agree, very closely demonstrating to my mind the fact that the

fourth stage when accurately determined indicates the true diastolic. I find, however, in three out of five of the leading hospitals of New York City, the fourth phase is established as the standard. The other two represent and are closely associated with medical schools. One adopts the fourth phase uniformly; the other holds strictly to the fourth phase on one division, whereas on the remaining divisions no definite rule exists, and part of the staff adopt the fourth and part the fifth phase as their standard. The last mentioned situation will, I believe, be found to exist in many institutions throughout the country.

The practical difficulties in routine life insurance or even clinical work preclude the use of the cardiograph at this time, and until further improvements in technique can be developed, greater uniformity in readings can be secured at the fifth than at the fourth phase. It represents an average difference of from four to eight points, which is only of importance where the reading is on the borderline of acceptance and declination. In such cases the allowance can be considered and action taken accordingly.

In order to confirm the above facts I have recently had eight of our experienced Home Office examiners report their findings after careful examination, with the result that in 35.8 per cent. of the cases the fourth phase was ill defined. The average diastolic pressure at the beginning of the fourth phase was 83.8 mm. and at the fifth phase, 77.4 mm., a difference of 6.4 mm. This represents a fair average and demonstrates the advantage of recording readings at the fifth phase from the standpoints of uniformity and accuracy.

The readings represented in Dr. Symonds' paper are of special value, owing to the fact that in the earlier years no readings were taken except by these carefully trained physicians. The combination of data covering age, weight and pressure in separate weight-groups, arranged according to the build-group of the Medico-Actuarial Mortality Investigation, is most satisfactory and must appeal to us all. The suggestion that in Table 2 there is a possibility that some of the marked

overweights are experiencing interference with heart action on account of the fat, seems quite likely. Table 3 certainly represents a fair average standard of systolic pressure for men. The résumé of the results of the four Companies outlined in Table 5 is instructive and the evident influence of war conditions upon the averages obtained, most interesting.

I am glad to note that in his reference to diastolic pressure in men, Dr. Symonds reports the same convictions which I hold regarding the superior practical value of readings taken just prior to the final disappearance of sound.

The suggestion of the possible influence of menstruation upon the pressures systolic and diastolic among women under 40 years of age is especially interesting to me and is I believe correct.

The pronounced variability of pulse pressure, viz.: from 20 mm. to 60 mm. in apparent health certainly indicates a definite question as to its value from the insurance standpoint.

The importance of careful search for evidence of aortic leakage and arteriosclerosis in the cases referred to in the reference to Table 16 is especially true, and should always be borne in mind when similar findings are recorded.

The suggestion that pulse pressures less than 30 mm. and greater than 56, should be regarded as a warning signal to examine the heart and vessels with great care is a good one and should be followed in practice.

I quite agree with Dr. Symonds in his conviction that the methods of obtaining blood pressure followed in insurance practice are both trustworthy and reliable.

In conclusion I wish to thank the Doctor for this valuable addition to our records.

Dr. L. F. MACKENZIE—Before discussing this paper we wish to express our sense of indebtedness to Dr. Symonds for his important contribution to our knowledge of blood pressure. The large number of cases entering into the analysis and the qualifications of the examiners ensure reliable results and place our knowledge of the normal on a more secure foundation.

The elaborate study by build-groups explains much of the rise in pressure at the older ages and heavier weights. Age, however, seems to be a considerable factor in its causation. We think that size and firmness of the arm also have an effect on the height of the pressure.

Dr. Symonds states: "It is not known what is the range above and below these averages which will give insurable mortalities." We would like to have the doctor tell us what are the limits above and below the average for age which he considers it safe to accept at standard rates. The Prudential, and we believe the New York Life and Northwestern Mutual, have been using 15 mm. above as the high limit acceptable for un-rated business. We feel greater leniency with reference to the low limit is permissible. Certainly the Mutual's mortality of 62.5% suggests increased liberality in our standards is possible if theirs have been the same. Dr. Fisher's recent investigations show, on the other hand, that blood pressures 10 to 14 mm. in excess of the average for the age gave a mortality of 136% by the new American Men Mortality Table. Such a percentage throws doubt on the advisability of considering at standard rates pressures as high as 15 mm. above the average.

Dr. Symonds thinks our average systolic pressure (125 mm.) too low owing to the large percentage of cases at the younger ages—there were 70.7% for ages 15-39 and 29.3% for ages 40-66—and that Dr. Fisher's 128.9 mm. is too high owing to the larger percentage being at the older ages. We have no doubt this is true and that the normal average for all ages lies somewhere between. Dr. Symonds' average for the younger ages is but 0.3 mm. higher than ours, with the age distribution practically the same—15-39 years—70.4%; 40 years and over—29.6%. His younger group was but 0.3 points smaller than ours. The differences between all the tables quoted and the doctor's are, however, very small, the greatest being 3 mm.

Our available blood pressure records from women are so few we have attempted no analysis of them. It was with particular pleasure, therefore, that we reviewed this part of the report under discussion. Generally accepted statements as to distinct

differences between the pressures of the two sexes are shown to have been incorrect.

We are in very hearty sympathy with the comments on the point at which to read the diastolic. The following table indicates improvement by the examiners and further justifies the use of the fifth point:

TABLE 1

FOURTH PHASE—ALL EXAMINERS

<i>Years</i>	<i>1912-1916</i>	<i>1922</i>
<i>Cases</i>	<i>1835</i>	<i>77</i>
<i>Mm. Hg.</i>	<i>%</i>	<i>%</i>
2-6	74.9	89.6
7-8	10.7	1.3
9-10	9.0	7.8
Over 10	5.3	1.3

The 1922 cases are few, but were examined by 19 doctors of different grades of efficiency. One of them who reported on 13 of the cases showed the phase as 5 mm. in eight and 10 in five. Sixty-nine of the seventy-seven had a fourth phase of less than 7 mm., and of the remaining eight 5 were examined by one doctor.

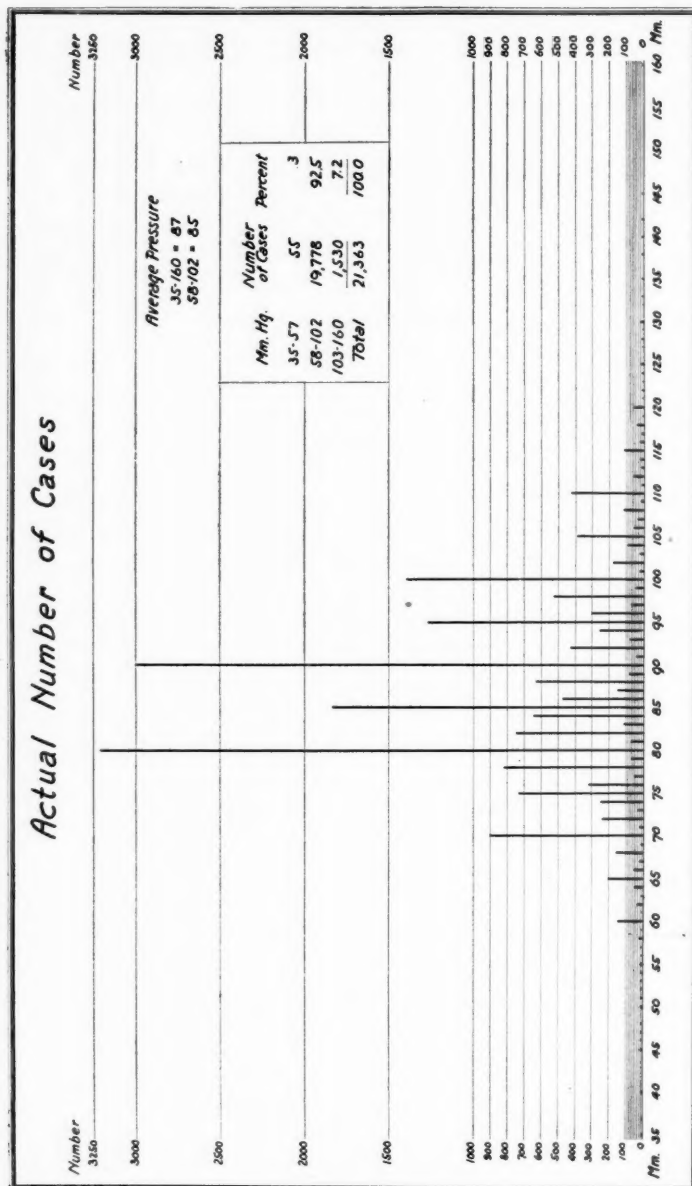
Here we wish to present six charts and a few tables to

TABLE 2

SYSTOLIC AND DIASTOLIC BY AGE GROUPS AND ALL EXAMINERS

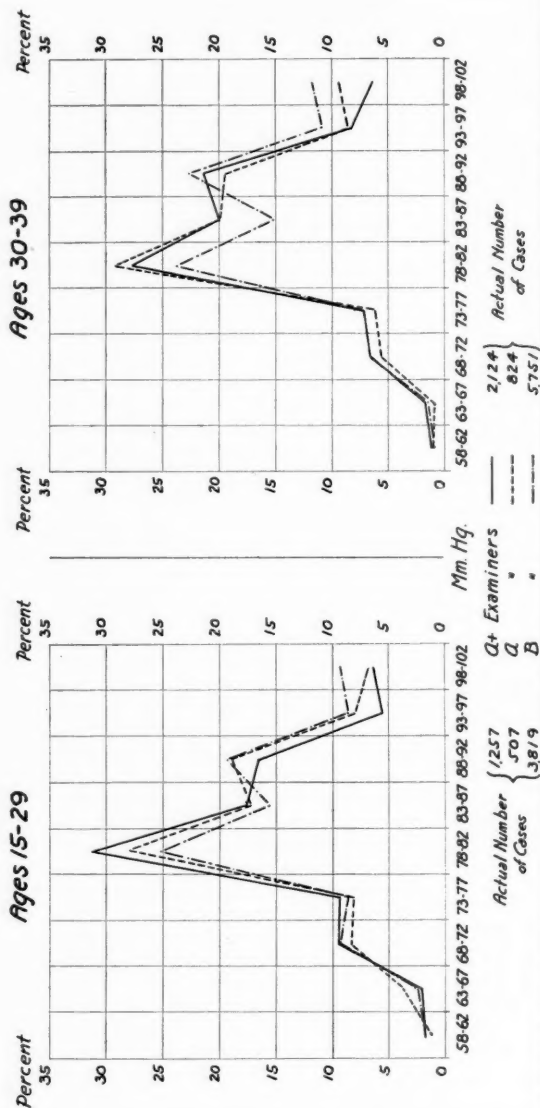
Age	Cases	Systolic	Diastolic	Pulse Pressure
15-19	40	115	76	39
20-24	224	122	77	45
25-29	428	122	78	44
30-34	554	124	80	44
35-39	477	124	80	44
40-44	329	126	82	44
45-49	153	126	80	46
50-54	92	131	83	48
55-59	17	131	80	51
60-64	6	136	86	50
65-66	2	146	88	58
15-66	2322	124	80	44

Diastolic Pressure of Males by Mm. Hg.



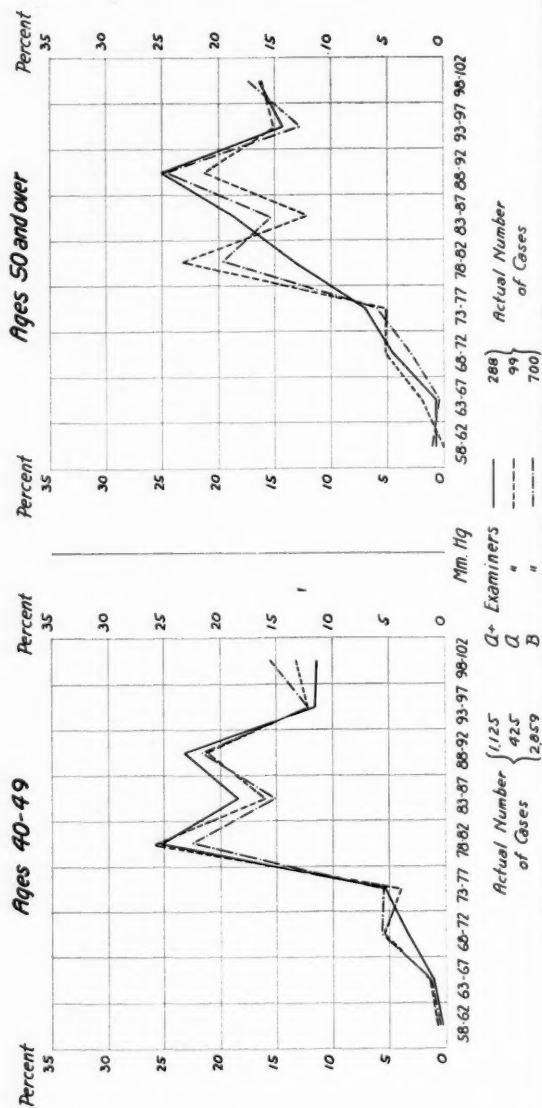
Diastolic Pressure of Males by Mm. Hg.

Percentage Distribution by Ages at Entry
and Grades of Examiners



Diastolic Pressure of Males by Mm. Hg.

Percentage Distribution by Ages at Entry
and Grades of Examiners



assist in explaining them, as these will aid in our further discussion.

The records in this table were from cases in which but one reading was made. The irregularities seen at the various age groups are probably due to the small number of cases. These records were derived from 2444 cases examined this year; the remaining 122 cases having had two or more readings.

CHART I

The actual distribution of the diastolic pressures recorded in 21,363 cases—the 1922 cases are included—is here shown. All were acted on at standard rates, with the exception of a few overweights. As we have always asked for the reading at the fifth point, this is the one represented here and throughout the charts. The readings ranged from 35 to 160 mm. It is very noticeable that the favorite points for taking the pressure are multiples of five, that even numbers are preferred to odd and that the pressures over 102 mm. (7.2%) were far in excess of those under 58 mm.—0.3%. The tendency in the past to read too high is apparent.

We have throughout used 58 to 102 mm. as the lowest and highest limits possible to consider normal. In this group there were 19,778 cases, or 92.5%.

The examiners were divided into three grades—A plus, A and B. The A plus grade is composed of our very best examiners, nearly all of them in large cities. The A group is a close second, while the B group is made up of all those not included in the other two.

CHARTS II AND III

These show by age groups and grades of examiners the percentage distribution of the cases.

The rise in the diastolic is well indicated in spite of the irregularities in and differences between the curves. That the different grades of examiners would today give more accurate readings will be seen by reference to Chart V. As in the age group

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50-66 the number of cases is small and very unequally divided among the grades, marked irregularities in and decided variations between the curves are noted. The curve of the A plus examiners in this group is different from the others, as there is a fairly smooth rise up to 90 mm. A slight upward bend at 80 and a downward bend at 85 mm. is, however, discernible.

CHART IV

Gives the percentage distribution by ages and A plus examiners.

The changes in the curves at the different age groups illustrate well, not only the increase in height of pressure with advance in years but also the tendency even amongst our best-qualified examiners to read too high and somewhat carelessly. The peculiarity of the curve of the oldest age group is more noticeable.

CHART V

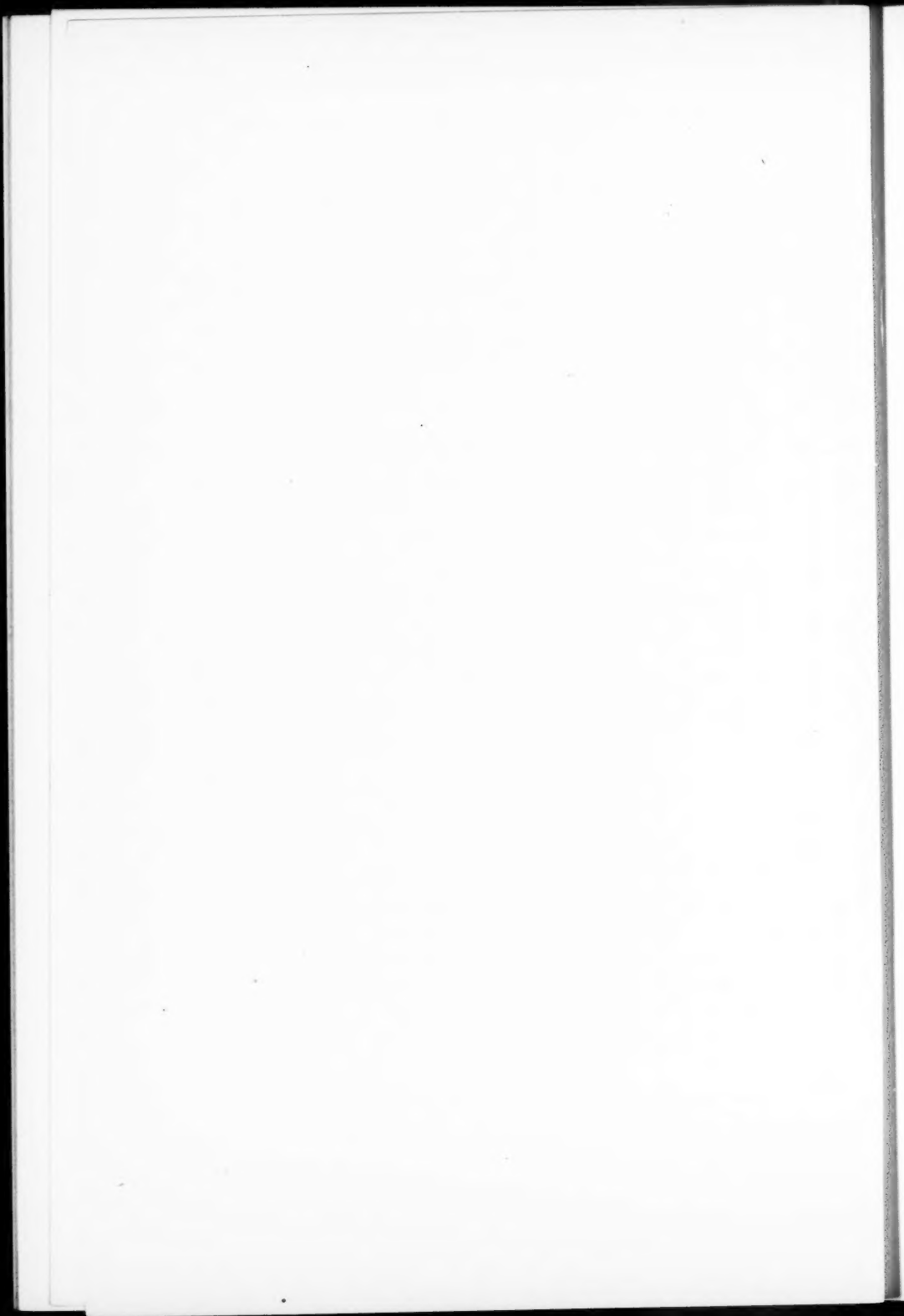
The percentage distribution for all ages by grades of examiners is shown. The cases were distributed as follows:

Grade A plus	4,794	
Grade A	1,855	
Grade B	13,129	
Total	19,778	100.0%
1922	2,444	12.4%
1912-1916	17,334	87.6%

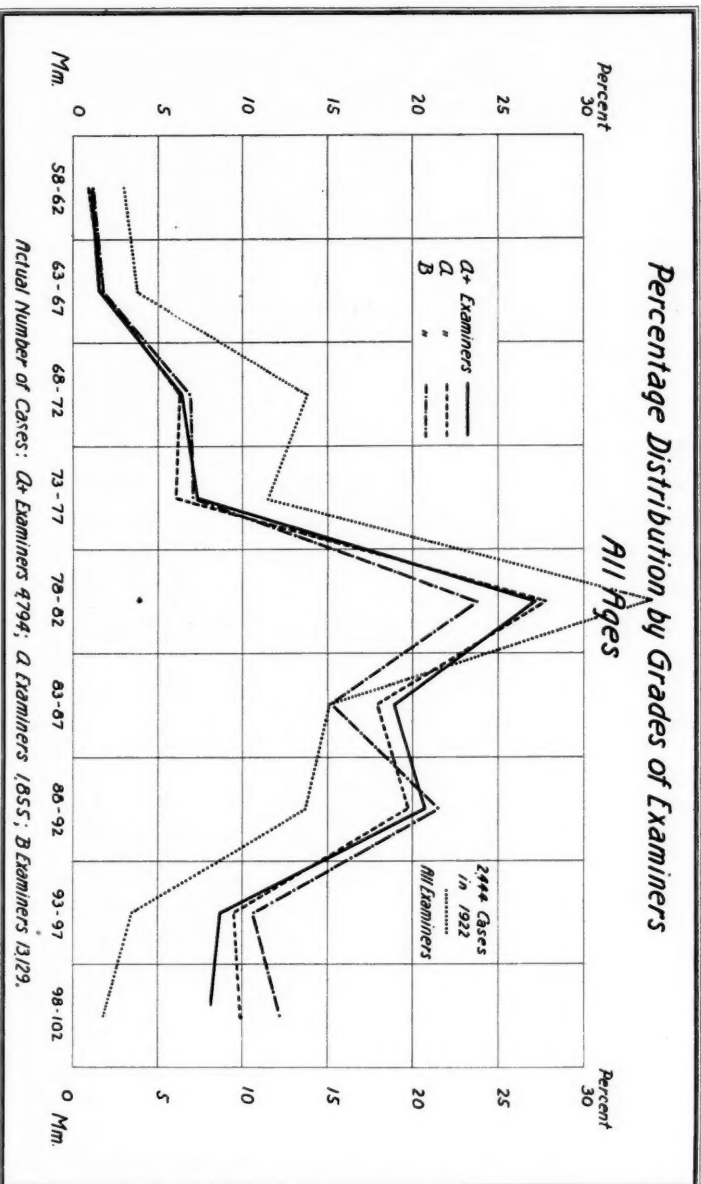
Even our best examiners had read too low up to 85 mm. and after that much too high. This is illustrated by the curve from the 2444 cases of 1922, which is superimposed on the others because of its smoothness and marked contrast to the curves of earlier records. There were just two cases more above than under 80 mm. (Table 4). We feel it points definitely to the more general accuracy of present-day readings, as the records of all and not selected examiners were used.

Percentage Distribution by Ages





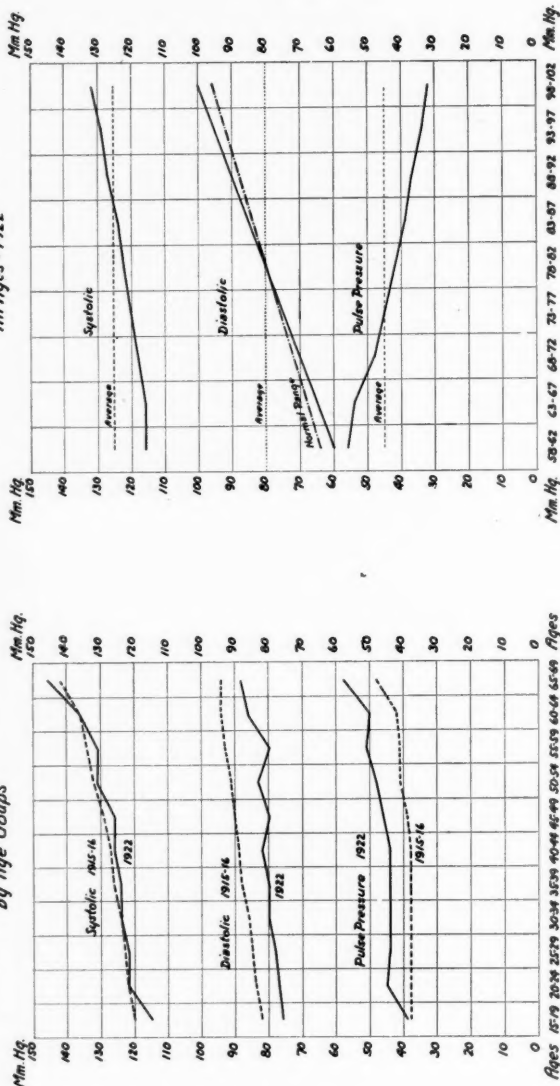
Diastolic Pressure of Males by Mm. Hg.



Blood Pressure of Males by Mm. Hg.

All Examiners

1922 Cases Compared with 1915-16 Cases
by Age Groups



Number of Cases 19,778

The Prudential Insurance Co. of America

We think it also supports our opinion as to the normal diastolic limits. It is derived from 100% of the 1922 records; the other curves from but 92.5% of 21,363 cases.

CHART VI

On the left side of this chart are shown graphically by age groups the systolic and diastolic for the periods 1915-1916 and 1922. The similarity of the two systolic curves is marked. The 1922 readings averaged slightly lower—2 mm. The fall in the diastolic is pronounced and averages 7 mm. The drop is most marked from ages 44 to 59. The rise after 59 means little, as there were but 8 cases. The pulse pressure increase is 5 mm.

On the right side are shown the systolic and pulse pressures based on the diastolic. The diastolic base line given is arbitrary and extends from 60 to 100 mm. The cards were thrown in consecutive order by mm. of diastolic, and the systolic averages for each 5 mm. diastolic group determined. The result is shown in:

TABLE 3

SYSTOLIC AND PULSE PRESSURES BASED ON THE DIASTOLIC. ALL AGES AND ALL GRADES OF EXAMINERS

Cases	Systolic	Diastolic	Pulse Pressure
211	116	58-62	56
334	116	63-67	51
1,336	118	68-72	48
1,394	120	73-77	45
4,922	122	78-82	42
3,237	124	83-87	39
4,179	127	88-92	37
1,990	129	93-97	34
2,158	132	98-102	32
19,761	125	80	45

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In the group tables from which this one was derived there was a variation of but 2 mm. between the grades of examiners at two diastolic levels. At the other levels there was no difference at two and not over 1 mm. at the remaining five. All grades were therefore combined.

The upward trend of the systolic as the diastolic rises is well shown. The change in size of pulse pressure is, however, far too great.

We have concluded from a study of Chart V and the following table:

TABLE 4
DIASTOLIC AVERAGE AND RANGE—1922

Cases	MM. Hg.	Average	Range
954	58-79	72	16
534	80	80	
956	81-101	88	16
2444			32

that the normal average is 80 mm., the high and low limits 96 to 64 mm. or less and the range not over 32.

The dotted and broken lines show the indicated normal average and normal range respectively.

Were 64-96 mm. used as the limits, it would make the pulse pressure more uniform in size. The systolic curve would scarcely be affected. We are very doubtful that the diastolic range will eventually be as large as that shown above. Our records are certainly approaching narrower limits, and as greater improvement in readings occurs these will very likely be much closer to the average than is indicated by Table 4.

The relationship of the pulse pressure to the diastolic is again well shown.

The improvement that has taken place is further shown by:

TABLE 5

PERCENTAGE DISTRIBUTION OF ACTUAL CASES. DIASTOLIC, A PLUS
EXAMINERS, ALL AGES, TWO PERIODS

MM. Hg.	1912-1916	%	1922	%
Under 58	11	0.2	0	0.0
58-102	4081	95.4	713	100.0
Over 102	188	4.4	0	0.0
Total	4280	100.0	713	100.0

There were 17 times as many cases over 102 mm. as under 58 mm. in the 1912-1916 period. In 1922 all were in the 58-102 mm. group. Only four of the 2444 cases in 1922 had readings above 96 mm., while 75 were below 64 mm.

Dr. Fisher in a personal letter tells us "Our experience at this office is that we find very few cases where the diastolic pressure registers more than 95 mm."

We believe that as time goes on the normal diastolic average will still be at or very near 80 mm., but high and low averages will more nearly approach the 70 to 90 mm. now frequently mentioned as the normal limits.

The earlier diastolic pressures of all companies seem to have been too high, even when read by the best examiners if the data given above is a true picture of what has happened. May there not then be a possibility that "the theoretical pressures for men" in Table 11 will later be found too high, especially in the younger age and lighter weight groups?

Table 15, "Home Office Cases—Pulse Pressure," leads us to think Dr. Symonds is in a position to set us right on this point if we are wrong. The records from which this table was derived are unusually valuable and very recent. A statement from the doctor as to the average diastolic pressure at the different age groups shown in this table and the average for all ages will be of great value and much appreciated.

Again, Table 15 shows a decided rise in pulse pressure after age 49, due to the fact that the systolic rises faster after this age than the diastolic. Comparison of Tables 2 and 10 shows an increase of 6.8 mm. in the systolic and 1.7 in the diastolic, a difference of 5.1 mm. in this age period. The same condition is shown in our Chart VI. As our diastolic pressures for 1922 are lower than those given by the Mutual, our average pulse pressure is a trifle larger, 44.3 when carried to the decimal (Table 2), as compared with 42.8, a difference of 1.5 mm.

The pulse pressure as pointed out by Dr. Symonds is not a real measure. It is the result of two variable pressures and subject to great changes in size. Even very large pressures may be due to temporary and functional disturbances and have no unfavorable significance. Change in size from variations in the diastolic is more significant of trouble than are equally great variations in the systolic.

We have considered 60 mm. the high limit of this pressure for standard insurance, but have been more conservative with the low limit as we believe it should be at least 25 mm. greater than the diastolic if the circulation is to be really efficient. Under 25 mm. we fear the presence of some unreported impairment is producing asthenia, general, or of the myocardium.

The systolic and diastolic pressures giving a pulse pressure of 20 mm. mentioned by the doctor are, of course, both well within normal limits, but the combinations as given, if received from Grade A plus or Grade A examiners, would certainly suggest to us the advisability of an additional reading and more information as to the applicant's physical condition.

In young applicants who are good risks in all other particulars, and who have a satisfactory diastolic, a pulse pressure of 70 mm., being undoubtedly functional, would not give us any uneasiness. Over age 35, however, we would want another reading and an explanation as to the probable cause of the high systolic at the first interview. The overload on the heart, if constant at any age, could hardly fail to result in hypertrophy.

Decreases in pulse pressure due to rise in the diastolic, or fall in the systolic, or both, following exercise, are, we believe,

very significant of circulatory or renal impairment, particularly if there is slow return to the original readings.

DR. WILLIAM R. WARD—One of the greatest services, if not the greatest service which this Association has rendered to the Science of Medicine, has been the study of blood pressure and the effect of abnormal blood pressure upon mortality.

In this study our Association and the entire Medical World are greatly indebted to Dr. Fisher whose persevering efforts covering a period of fifteen years have given to us such a wealth of valuable information.

In 1907 Dr. Theodore C. Janeway published his classic entitled "The Clinical Study of Blood Pressure." That date marked the beginning of a new epoch in medicine, and especially in Life Insurance selection. Some of the Insurance Companies were alert in utilizing this new method of diagnosis and consequently they required a blood pressure reading in applicants over forty years of age when the amount was \$25,000 or over. In October, 1909, Dr. Fisher in discussing the subject of rheumatism informed the Convention that the Northwestern had used the sphygmomanometer in certain cases since 1907 and that they had declined 139 applicants because of a high pressure. At that session Dr. Rogers remarked: "We shall all be using the sphygmomanometer as a routine procedure just as we have for some years examined the urine as a routine procedure."

In 1911 Dr. Fisher had accumulated sufficient data to present to us his comprehensive report upon the use of the sphygmomanometer, and since that session this subject has had a prominent place in every meeting.

It is both interesting and instructive for us to review at this time some of the facts made known to us in 1911.

At that time the blood pressure in applicants under forty years of age was considered of relative unimportance, but Dr. Fisher stated that 446 applicants under forty years of age had been examined at their Home Office and that twenty-six had shown a pressure exceeding 150 mm. In his present report he states that 743 applicants under thirty years of age were de-

clined because of a high systolic pressure and no other impairment, and that the mortality of this group was 195.7%; also that 731 applicants between thirty and forty years of age were declined because of a high systolic pressure with no other impairment, and that their mortality was 259.5%. There can no longer be any question as to the significance of an abnormal pressure in applicants under forty as well as in the middle life period.

Another most interesting feature of that 1911 report is the average pressure as tabulated at that time.

If we compare this table with the recent and exhaustive study made by Dr. Symonds, we observe that the early table showed a somewhat higher average pressure at every age period. We must also bear in mind that the pressures then recorded were taken by the palpatory method whereas a large proportion of the pressures comprising Dr. Symond's table were taken by the auscultatory method. Why this difference? Possibly the fact that in the early days a narrow band was employed, may have made some difference,—then too, at that time the Examiners may have recorded the pressure as they first found it, whereas today, if the pressure is rather high at first reading, an Examiner may make several subsequent tests and then report the average.

In Dr. Fisher's present report he shows us that 4165 risks rejected for high systolic pressure alone give a mortality of 230.5%. In reviewing this table we note that the risk in these cases is not a deferred risk, but that the mortality is very high during the first year and also during the first five years as well as during the subsequent years. He directs our attention to the fact that in Table 2, 525 cases with an average systolic pressure of 12 mm. above the average, gave a mortality of 136.1%.

This is rather disconcerting information for those of us who have believed that applicants with a pressure not exceeding 15 mm. in excess of the average were on a safety zone, and does not harmonize with the mortality of the 520 accepted risks whose blood pressure averaged 153 mm., which was considerably more than 12 mm. above the average.

Dr. Fisher's explanation for this difference is that a majority of these accepted risks were approved with only one reading and that consequently the actual pressure may have been considerably lower than the pressure as stated. Furthermore that forty-eight cases which had lapsed, etc., and whose conditions were unknown, were treated as still living.

Even with this explanation, it is difficult to understand the difference between the mortality of the accepted risks and the declined risks—there having been no other impairments. Is it not probable that in the accepted risks with hypertension, many of the cases were so far above the average in other respects that they were approved notwithstanding a rather high pressure, whereas in the declined risks although there was no other actual impairment the cases may have lacked those other superior qualities which warranted the acceptance of the approved risks. We must also bear in mind the fact that in this group of 525 declined risks with an average excess pressure of 12 mm., there were only 26 deaths. Because of this fact I have asked Mr. Papps, our Mathematician to express an opinion from an actuarial standpoint. He has made the following comment.

"In any mortality statistics involving comparatively few lives, and particularly where there are few expected deaths, a great deal of caution should be used in drawing any conclusions. In Dr. Fisher's paper he quotes the results of 525 cases where there were 19 expected deaths and 26 actual. This gave a percentage of 136.1. With this he contrasts the mortality of 520 cases where the ratio of actual to expected was 114.4%. In looking up the previous reports of the Committee, it would appear that there were actually 532 cases with 80 expected and 94 actual deaths, showing a percentage of 117.5. Coming back to the 525 cases with 19 expected and 26 actual deaths, the mortality percentage is 136.8. Presumably the expected deaths were a fraction over 19. Assuming, however, an even number of 19 expected deaths and 22 instead of 26 actual deaths, the mortality percentage would have been 115.8. A variation of four deaths, which might readily arise from acci-

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dental causes, would produce a mortality in the 525 cases less than in the 532. This shows that the variation of a very few deaths will very materially alter the percentages where so few deaths are involved. To take an extreme case, if there are but two expected deaths and two actual, we have a mortality of 100%. One more actual death would raise the percentage to 150. On the other hand, if there are 100 expected and 100 actual deaths, one additional actual death would increase the percentage by unity instead of by 50."

The Mutual Benefit now allows a pressure of 15 mm. above the average. The practical question that now confronts us is, shall we change our present rule to a lower margin, say 10 mm. or 12 mm. so as to avoid this excess mortality. Speaking for the Mutual Benefit, I do not think that it is advisable to do so. Our mortality experience for 1922 is under 50% of the expected and it does not seem to us necessary to reduce the mortality still lower.

From this report of Dr. Fisher and those that have preceded it, it is obvious: (1) That the blood pressure should be procured in every case irrespective of age or amount. (2) That a low pressure does not constitute an insurance hazard. (3) That a constant pressure in excess of 12 mm. above the average should at least be viewed with caution and that the tendency of the most competent observers is to reduce the normal zone of hypertension rather than to increase it.

Dr. JOHN W. FISHER—Dr. Symonds, as usual, has furnished an abundance of valuable data on the subject under discussion. I infer from his statements that he has included the palpatory readings prior to the year 1915 with those of the auscultatory readings since that date. What is needed is a comprehensive table of the average auscultatory blood pressure at the different ages. The averages of 150,419 entrants, as shown in Table 2, varies but slightly from the averages compiled by the Prudential, New York Life, and a combination of the Mutual Benefit and Northwestern, which were almost entirely based upon the palpatory method; except, possibly, that of the New

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York Life, which shows practically no variation from the MacKenzie and Fisher averages at any of the group ages.

SYSTOLIC BLOOD PRESSURE—MEN ONLY

AGE	AVERAGE					
	Fisher	MacKenzie	Hunter and Rogers	(1)	Symonds (2)	O Build (3)
15-19	120	119	120	121.	121.2	123.4
20-24	122	122	122	123.	123.4	124.3
25-29	123	123	123	123.4	123.9	124.4
30-34	124	124	124	123.1	123.8	123.9
35-39	126	126	125	123.9	124.9	124.4
40-44	128	127	127	125.	126.5	126.2
45-49	130	129	129	125.9	128.4	127.4
50-54	132	132	133	127.7	130.9	129.8
55-59	134	135	134	129.7	133.9	133.1
60-64	135	137		129.8	135.2	135.1
65 and Over	136	139				

Column (1) in the Symonds, data represents the average systolic blood pressure of those 140 mm. and below, as shown in Table 4.

Column (2) represents the average systolic blood pressure, as shown in Table 2.

Column (3) represents the average systolic blood pressure, Build o, as shown in Table 2.

Dr. Symonds' data is in splendid shape, no doubt, to furnish us with an accurate auscultatory systolic and diastolic average pressure at different age groups. By discarding all of the palpatory readings, or, what is better, compile a palpatory average and an auscultatory average, he would have an abundance of material in the issues of any two years; namely, 1915 and 1916, or 1918 and 1919, or the four years combined. He now has an accurate table, no doubt, of the average diastolic pressure.

It is barely possible that we may find that blood pressure

averages are somewhat lower since 1915. We have some such evidence in our examinations at the Home Office, and this point is being investigated.

I would suggest to Dr. Symonds that he compute the mortality of the 3037 cases in which the systolic pressure is over 140 mm. at ages 15 to 39. Almost 3 per cent. of these ages, in Table 2, show a blood pressure of over 140 mm., which is, except at ages 35 to 39, more than 15 mm. over the average for the age; while at ages 35 to 39 the blood pressure is 14 mm. above the average, and a compilation of the mortality of 4186 cases in which the diastolic pressure is over 94 mm., provided his records are in such shape that he has positive knowledge that the pressure in both groups was persistently high, as shown by repeated readings. Possibly the Doctor has this in mind and has made the compilation, as he intimated in his correspondence with me that he hoped to have some additional statistics along this line.

In our Home Office examinations during the past two years we have determined the palpatory and auscultatory readings and also the diastolic reading at the last loud tone and also at the cessation of all sound, and find there is an average of about 5 mm. between the palpatory and auscultatory systolic and an average of about 4 mm. between the last loud tone and the cessation of all sound in the diastolic. However, as we have had only about 1500 cases, we are not prepared to state positively that this is a correct average variation.

From our experience thus far I am inclined to the belief that the high diastolic pressures, as a class, do not apply for standard insurance; that when the subject reaches the stage of high diastolic, he has reached the stage which suggests a consultation with his medical adviser. That is, he has developed, possibly, some form of cardio-vascular-renal symptoms. It is a well-known fact that in the onset, as a rule, of hypertension, there is a feeling of well being, so that he can see no reason why he should consult his medical adviser, and will not as a general rule carry out his instructions should he consult him, and this is just the period that proper treatment is of advantage. This

fact is well known to the alert clinician. In this I do not contend that we should not endeavor to learn all that is possible for us to discover as to the significance of the diastolic blood pressure, especially as it effects risk selection.

We have in our work furnished the general practitioner with valuable information concerning the systolic blood pressure, and should do all in our power, especially through the local examiner, to persuade the rejected applicant to seek medical advice in all cases of high tension coming under our observation, and to impress him with the advantage of carrying out the instructions of his medical adviser, notwithstanding he may feel that he enjoys perfect health. Much can be accomplished along these lines with those companies which issue substandard insurance to this class.

Dr. Symonds—Mr. President and Gentlemen: Dr. McKenzie asked in his discussion what the limits were above and below the average for age at which it is safe to accept risks at standard rates. I am not prepared to answer that question from the meager information which we have. In the older ages there was too much tendency, at that time, to exclude low systolic, and furthermore in regard to the high systolic I think that we have to reconsider perhaps some of our views on its significance in relation to average. The average goes up and the mortality among those who are averaging high also goes up, so that in other words the normal is probably not coincident in rise with the average and I think we will have to look out for those higher blood pressures in the later ages. On the other hand, in the younger ages the low systolic pressures have a tendency, as shown by the limited experience that I have given in my subsequent report to show a rather high mortality. The low systolic in the young ages is associated rather with light weights and whether it is the light weight or the low systolic that is perhaps an indication of a little lower general tone, I do not know, but the mortality does run a little high among those, while among the oldest ones the low systolics seem to be very good risks.

I think Dr. McKenzie took a little to heart the comment I

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made in my paper. I said that Dr. Fisher's general average was a little high and that his was lower. His is only .3 mms. lower than ours, and I think the results set forth by Dr. McKenzie in his paper two or three years ago were very excellent. Dr. Fisher's general average was high because he did not have many in the younger ages.

The spread of the fourth phase is very interesting, as set forth by Dr. McKenzie. He says that now practically 90% of his Examiners report a spread of only 2 to 6 mms., covering the entire fourth phase. In his figures on the 9 and 10 mms. apparently there were six cases reported in that group, five of them by one man, so that I think almost all of those could be thrown out. The probabilities are that in 95% of the diastolic readings taken by a careful examiner, the spread of the fourth phase between the fourth and fifth point would be within 6 mms. In his table he noted that the favorite points for taking the pressure are even numbers and multiples of five, but much more so are multiples of ten. Multiples of ten stand out like a rag on a sore thumb, but the fact that the examiner favors even numbers is partially due to the apparatus. I think nearly all of the manometers are graduated only in even mms., that the odd mms. are omitted and the tendency of the examiner is to read the even mms.

Dr. McKenzie's table No. 3 shows undoubtedly a very high correlation between the systolic and the diastolic. That in fact is borne out by the pulse pressure itself which runs along at forty-two up to nearly fifty years of age. Unfortunately, of course, there are cases in which the correlation does not work and those are the exceptional cases which you have to take into account.

Now he asks a question with regard to the diastolic pressure in recent years, and I am glad to give him our experience at the Home Office.

Our blood pressure readings at the Home Office were taken from 1917—a few in that year—to June, 1922. The general average of the diastolic pressure was 82 mms. Dividing them up into years of issue:

Year	Entrants	Diastolic Average
1917	645	81.5
1918	880	82.1
1919	1071	82.1
1920	1202	82.3
1921	1338	82.6
1922 (first 5 mo.)	580	80.6

I do not believe that the diastolics are going to be modified very much in the future. They will run 80 to 82 pretty closely. I do not think there will be much of a reduction. The last group was a trifle lower than the others but the number is considerably smaller and there might well have been a little accidental variation.

Dr. Ward spoke about palpation, the taking of the systolic pressure by palpation and auscultation. Prior to 1915 a good many of our reports were undoubtedly by palpation, but after all they formed a rather small element in the 150,000 cases that I reported. Very many more came in in the years 1915, 1916, 1917, 1918 and 1919, and those were nearly all taken by auscultation. The diastolic was determined by auscultation.

The low systolics, I think, should be examined with care but outside of that if we get low systolics they are still good risks if otherwise they are sound. I think a low systolic ought not to bar us from acceptance.

Dr. McMahon—Dr. Ferguson has very kindly prepared a paper on Epilepsy in its Relation to Life Insurance, which will now be presented. I regret very much that Dr. Ferguson is not here himself to listen to and take part in the discussion of his paper.

EPILEPSY IN ITS RELATION TO LIFE INSURANCE

BY JOHN FERGUSON, M.A., M.D.

The Excelsior Life Insurance Company, Toronto

When Dr. T. F. McMahon requested me to prepare a paper on this subject, I felt that his request was almost a command;

for in years gone by, fast receding into the past, he was one of my esteemed students in the Department of Anatomy, but now the worthy President of the Medical Directors Association of America, while I am but a private under his captaincy. I therefore gave him my promise to prepare a paper on the topic suggested.

At the commencement I may state that I hold no brief for or against the victims of epilepsy. I shall approach the question of the insurability of epileptics with as judicial a mind as I can command, trusting that the more experienced members of the Association shall fill in the many blanks to be found in my paper, and complete what I may leave unfinished. A great writer once said that it is through gropings we arrive at the truth, and by the collating of experiences that we perfect our knowledge. If my paper, and more especially the discussion thereon, shall aid in clarifying our views and finding a basis for action, the hour shall be profitably spent.

The late Sir W. R. Gowers, a much loved teacher, tells us: "The characteristic of the malady is the recurrence of sudden brief disturbances of some functions of the brain, varying in degree, extent, and character, but generally attended with an arrest of consciousness sufficient at least to interrupt the control of the muscles necessary for maintenance of the erect posture." Dr. W. Aldren Turner, an eminent authority on epilepsy, defines it thus: "Epilepsy is a chronic progressive disease of the brain, characterized by the periodic occurrence of seizures, in which loss of consciousness is an essential feature; commonly associated with convulsions, and frequently accompanied by psychical phenomena of a well defined type; occurring generally in persons with a hereditary neuropathic history, which shows itself in signs or stigmata of degeneration; running its course uninterruptedly, or with remissions, over a number of years; and terminating either in a cure, in the establishment of the confirmed disease, in delusional insanity, or in dementia."

Dr. L. Pierce Clark in Wood's *Reference Handbook* defines the malady thus:

"Epilepsy is a chronic disease of the brain characterized

by convulsions and loss of consciousness, either one of which may be absent in exceptional instances. There are all possible gradations of the disorder of consciousness, from complete loss to that of complete retention of it. A complete loss of consciousness for a longer or shorter period is the general rule. The muscular spasm of epilepsy is similarly subject to variation, but is much less frequently absent than loss of consciousness. The manifestations of the disease in seizures or attacks have usually been classified according to their severity, such as grand mal, petit mal, and psychic epilepsy. The first is usually characterized by the severe attack, which ordinarily consists of loss of consciousness and great violence of muscular spasm, while the second is shown in a slight seizure with partial or incomplete loss of consciousness and but slight muscular spasm. Psychic epilepsy is that manifestation of the disease shown in the maximum of mental confusion, and more or less complete loss of consciousness without obvious convulsion."

The disease was given its name epilepsy by Hippocrates. Before passing to the aspects of the disease that concern us today, let me quote a few lines from the Latin poet Lucretius, as translated by Dr. Mason Good:

"Oft, too, some wretch, before our startled sight, struck as with lightning, by some keen disease, drops sudden. By the dread attack o'erpowered. He foams, he groans, he trembles and he faints; now rigid, now convulsed, his laboring lungs heave quick, and quivers each exhausted limb. He groans since every member smarts with pain, and from his inmost breast, with wontless toil, confused and harsh articulation springs. He raves since soul and spirit are alike throughout distracted by the bane. But when at length the morbid cause declines, and the fermenting humors from the heart flow back, with staggering foot first treads, led gradually on to intellect and strength."

The reading of these four descriptions of epilepsy would be apt to lead one to the conclusion that the epileptic would prove a doubtful subject on which to hazard one's money when duration of life must determine whether the result shall be

loss or gain. I hope, however, to show that the epileptic has some claims to our consideration, and that he is not wholly without credit marks; but it will also become clear that much caution must be observed in making selections from this group.

One of the first questions to be considered is the number of epileptics in the general population of a country. The most conservative observations have agreed in estimating that in the United States and Canada there is one epileptic in every five hundred of the population. This would give about 220,000 in the United States, or 150,000 in the registration areas covering a population of 75,000,000, and for Canada at least 19,000 epileptics. These numbers are sufficient to warrant insurance experts in giving due attention to this group of the general population, and in endeavoring to ascertain whether or not there are any in the group to whom insurance may be safely or justifiably granted.

Epilepsy is essentially a young people's disease. Fully 80 per cent. of all the cases commence by the twentieth year; and at least 85 per cent. by the twenty-fifth year. The reasons for this become apparent when one looks into the etiology of the malady. In the first place heredity plays a most important part in causation. Gowers states: "We may safely conclude that in at least 50 per cent. of all cases of epilepsy the malady is ultimately the result of neurotic inheritance." W. A. Turner from a study of his private and hospital cases, puts the hereditary influence at 51 per cent. Spratling, in his book on epilepsy estimates this factor at 56 per cent. W. T. Shanahan, medical superintendent of the Craig Colony for Epileptics, at Sonyea, N. Y., with his ample opportunities for making correct deductions, is of the opinion that hereditary influences can be traced in from 60 to 80 per cent. of all cases. Prof. C. W. Burr, in the *Archives of Neurology* for June, 1922, states that of 1449 cases he found a direct or indirect neurotic history in 621.

These figures go to show why epilepsy is so peculiarly a disease of the young; for, in the vast majority of instances, hereditary tendencies reveal themselves during the years of growth and development of the body and mind. But there are other

reasons for this also. Whatever the heredity may be, there are sound reasons for believing that injuries to the brain in its early stages of growth play a most important part. W. T. Shanahan puts the case thus:

"To sum up many of the etiological factors, epilepsy may be considered as organic in a broad interpretation of the term, namely, fetal encephalitis and meningitis due to hereditary syphilis or some other destructive agent acting during intra-uterine life; obstetrical or subsequent trauma to the brain and its membranes; scarlet fever, measles, typhoid fever, etc., complicated by inflammatory processes affecting the central nervous system; cerebral endarteritis, etc., due to acquired syphilis, lead intoxication, etc.; cerebral neoplasms; cerebral hemorrhages during paroxysms or pertussis; sunstroke; arteriosclerotic changes in cerebral vessels; changes due to chronic alcoholism, endocrine maladjustment at puberty, and at the involutional period, etc. These epilepsies, strictly speaking, are symptomatic."

As we acquire information the number of cases of the so-called idiopathic epilepsy become fewer. With the language of E. B. Block in Tice's *System of Medicine*, most will concur:

"Anybody who is looking for one cause of epilepsy might as well stop looking. Epilepsy is a symptom of many disorders, organic, physiological and chemical. It seems evident that epilepsy is not due to one cause, but to a combination of causes, which are necessary to produce the attacks." The same writer gives this definition: "Epilepsy is a disease in which there occur repeated transient attacks of either a psychic sensory, or motor nature with a loss or impairment of consciousness." This definition admits of great width of application.

W. A. Turner, in his Morison lectures, divides Epilepsy into the following groups:

1. The organic epilepsies, (a) Those forms from injury to the skull, brain, or membranes. (b) Those from focal or organic disease, as a tumor or thrombosis.
2. Epilepsies beginning in childhood or infancy. There is usually imbecility or idiocy present.

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3. The epilepsies of later life such as (a) cardio-vascular disease, (b) intoxications, (c) from dementing psychoses, (d) those arising from eclamptic conditions as uræmia, etc.

4. Idiopathic epilepsy from some chronic disease of the brain the nature of which is not known, and characterized by seizures with loss of consciousness as the essential feature and eventually leading to more or less impairment of mind or dementia.

With regard to the proportion of cases that are organic and idiopathic, the following table, taken from the latest report of the Craig Colony, is instructive:

<i>Organic 121</i>	<i>Male</i>	<i>Female</i>
Fœtal Mal-development.....	21	8
Birth Trauma.....	7	1
Subsequent Trauma.....	10	7
Encephalitis and Meningitis.....	6	15
Syphilis.....	2	1
Alcoholic.....	2	1
Arteriosclerotic.....	5	3
Neoplasms.....	1	0
Sunstroke.....	1	1
Infections.....	7	0
Metabolic.....	1	0
Endocrine.....	11	10
Total.....	74	47
<i>Idiopathic 155</i>		
Psychogenic.....	11	9
Hereditary.....	19	20
Unclassified.....	52	44
	82	73

It is quite fair to presume that many of the unclassified cases in the idiopathic group would belong to some of the divisions of the organic group were all the facts of etiology known. Redlich thinks the term idiopathic should be abandoned and holds the disease to be organic, though the morbid anatomy not yet discovered.

E. B. Block in Tice's *System of Medicine*, Vol. X., page 406, states: "The causes of idiopathic epilepsy are not evident on examination. Sometimes these cases are due to jars or jolts to the nervous system, concussion, or to small adhesions of the membranes, to small cerebral hemorrhages or localized

encephalitis, none of which gives definite evidences at the time of examination on account of the development of epilepsy. Idiopathic epilepsy is rapidly being reduced in frequency by more careful study of the cases."

On the matter of the age of onset, the following table prepared by W. A. Turner, gives very precise information.

Age	Total	Males	Females	Per Cent.
Up to 5	170	104	66	17.
6-10	143	76	67	14.3
11-15	281	150	131	21.1
16-20	186	106	80	18.6
21-25	69	31	38	6.9
26-30	53	37	16	5.3
31-35	34	20	14	3.4
36-40	24	12	12	2.4
41-45	18	9	9	1.8
46-50	8	7	1	.8
51-55	5	2	3	.5
56-60	5	2	3	.5
61-70	4	3	1	.4
	1000	559	441	100

From the foregoing figures it will be seen that 85 per cent. of the cases occur by the age of 25 years, and only 15 per cent. after this age.

Opinions have varied much as to the incidence of the disease in the two sexes. A careful review of large numbers makes it clear that the disease more frequently affects males than females. Gower's contention that young girls suffered more frequently than boys has not been borne out by statistics, as per this table.

Age	Males	Females
Up to 10	180	133
11-20	256	211
21-30	68	54
31-40	32	26
41-50	16	10
51-60	4	6
61-70	3	1

The deaths from epilepsy in the United States, as per the data for 1917, throughout the registration areas, reveal the following ratio of males to females.

Age	Males	Females	Age	Males	Females
Under 5	124	90	55-59	100	63
5-9	62	55	60-64	79	57
10-14	96	63	65-69	71	43
15-19	171	125	70-74	52	33
20-24	183	116	75-79	31	25
25-29	175	110	80-84	13	11
30-34	163	114	85-89	11	8
35-39	199	109	90-94	1	0
40-44	159	94	95-99	0	0
45-49	125	77	100 +	0	0
50-54	83	60	unknown	8	2

It is a fair assumption that the numbers suffering from the disease are in proportion to the numbers in the two sexes whose deaths were due to it. In the same year the total male epileptic deaths in the United States were 1906, and the total female epileptic deaths 1255.

Still further dealing with etiology it is well to recall the words of Gower's. "Epilepsy and insanity are interchangeable in families; they are certainly correlated." He then refers to hysteria, migraine, and dipsomania as having relationships to so many morbid states that they cannot be linked so closely with epilepsy as insanity. Dealing with the immediate cause he states: "The exciting cause of the disease is that of the first fit, because the malady is self-perpetuating. This first fit leaves behind it a tendency to recurrence which is often an increase in the primary disposition. For the most part the immediate cause is trifling, and the remote cause, the predisposition, is incomparably the more important of the two." W. A. Turner puts causation in these words: "It is held to be a cardinal principle, that the cause of epilepsy is that circumstance to which the first fit is apparently due." He goes on to state "that the epileptic tendency is a sign or stigma of a neuropathic inherited disposition, the anatomical basis of

which is seen in certain well-defined structural peculiarities both of the body and the cerebral cortex. In those who have inherited the epileptic tendency the convulsive habit may be established in the course of natural development, or as the result of occasional or accidental causes, such as injuries, digestive derangements, diseases of various organs, emotions, and many reflexes."

The following table gives the classification adopted at the Craig Colony.

CLASSIFICATION BASED ON ETIOLOGY

		(Porencephaly (Foetal)
		Birth or subsequent head traumas
		Encephalitis
		Meningitis
		Syphilitic { Endarteritic (Congenital)
		{ Encephalitis, etc. (Acquired)
		Alcoholic
		Arteriosclerotic
		Lead
		Neoplasms (Tumor—Gumma, etc.)
		Sunstroke
Organic (Symptomatic)	Infectious	{ Scarlet fever
		{ Typhoid fever
		{ Measles
		{ Pertussis (Mechanical?)
		{ Intestinal abnormality of function
		{ Uremic
	Metabolic	{ Eclamptic
		{ Diabetic, etc.
		{ Endocrinopathic { Involution
		{ Puberty
Idiopathic or Essential	{ Psychogenic (epileptic constitution)	
	{ Unclassified	
Allied	{ Paresis	
	{ Dementia Præcox	
	{ Spasmophilia	

CLASSIFICATION BASED ON SYMPTATOLOGY

Major—Convulsions (Grand Mal)	{ Complete
	{ Partial
Minor—Petit Mal (Mild) (With or without automatism)	
Psychic—Brief—Protracted—Recurrent	
Jacksonian—Localized Epilepsy	
Sensory—The varieties of Auræ	

MENTAL CLASSIFICATION

Normal (No. psychosis)

Insane

Feeble-minded	{	Moron
(Amentia)		Imbecile
		Idiot

Deterioration—Dementia—Enquire reoriginal mental status.

This table should be carefully studied by every examiner. It will aid him in understanding and recognizing the disease; and will enable him to furnish the Head Office with such information as would enable it to arrive at a just conclusion.

Percy Sargent, in his presidential address before the Section of Neurology, Royal Medical Society, January, 1922, gives weighty reasons in support of the vascular causation of epilepsy. Leonard Hill once taught that the vessels of the brain were not subject to vasomotor influences. This has been completely disproved. Sargent holds that a sudden contraction of the cortical vessels causes loss of inhibition over the lower centers. The first causes loss of consciousness, and the latter the tonic spasms. Presently as the blood supply of the brain is being irregularly restored there ensues the clonic spasms, a result of cerebral cortex irritation. Jacksonian epilepsy is limited to cortical irritation, hence there are only clonic spasms and, sometimes, no loss of consciousness, the storm not being widely enough distributed to cause such. Some emotion, reflex, pressure, irritation, toxic substance, or perverted endocrine action, is capable of suddenly blanching the cortex, and the chain of events is set into motion. In the Jacksonian type, enough of the cortex is not involved to liberate the lower centers from the control of the higher centers. This thoroughly accords with my own views.

In the same address, Sargent points out that in 18,000 cases of gunshot injuries to the head during the great war, 800 developed epileptic fits, or four and one half per cent. In a list of 270 cases of cerebral tumor no fewer than 82, or 30 per cent., suffered from fits of a focal character. There are many examples of fits resulting from cerebral hemorrhages, localized infections, meningeal adhesions, and injuries without gross lesion.

When one enters upon the study of the pathology of epilepsy, very great difficulties are encountered, as there appears to be a wide divergence of views. With regard to the cortical cells several changes have been observed, such as reduction in number, alteration in shape, loss of the dendrites, large or small nucleus, cells shrunken, vacuolation, and sometimes the cell is markedly swollen, indeed, the marks of degeneration. With regard to the membranes there is usually some fibroid thickening. Endarteritis is not uncommon, and the walls of the vessels reveal sclerotic changes. Several types of deposits are found, as leucocytes, red cells, fibrin threads, and Mulberry-like masses. The vessels are sometimes obliterated. Lying between the membranes there is a characteristic foam-like or frothy exudate. It contains no nuclei and is obviously an unorganized structure representing an albuminous exudate from the vessels. This exudate obstructs the lymph spaces, interferes with the nutrition of the tissues, and its fatty disintegration sets free injurious poisons. This is one of the most constant morbid changes found. Another change found in the brains of epileptics is an increase in the glia tissue and cells.

This neurogliosis is most pronounced in the surface layer of the cortex, but may involve its entire thickness. This overgrowth of neuroglia may form definite buds. The increase in the glial cells is met with most markedly in cases going on to dementia. These cells are usually small, but some are giant in type and contain several nuclei. This condition of gliosis was found in 50 out of 95 cases, or 52.6 per cent., by Turner, Clark and Prout, and Bleuler. The best authorities claim that the cornu ammonis is sclerosed in about 50 per cent. of the cases; but most regard this as a late condition in the disease; or as part of the general tendencies to sclerosis in the central nervous system of epileptics. It should be noted that sclerosis of Ammon's horn is extremely rare in those who were not subject to epilepsy. The thalamus is found to present atrophic changes, sooner or later, in epileptics. This seems to be similar in origin to the changes in Ammon's horn. Angiomas occur among epileptics much more frequently than among non-epileptics.

The cerebellum is almost invariably small, and, when carefully examined, is found to be studded with atrophic patches. The foliæ are wasted, there is an abundance of the foamy material already mentioned, and the cell elements are much reduced. Degenerative and sclerotic changes are found in the medulla and cord. These may be somewhat the result of changes in the cerebrum and cerebellum. Cholin is found in the spinal fluid of epileptics, but not in that of hysterics or neurasthenics. The many changes described are held by some to result from the vascular changes and the convulsions.

The morbid conditions that have been mentioned all go to prove that there are profound differences in the central nervous system of epileptics, as a class, to what pertains in those free from the malady. But there are many epileptics whose brains reveal no apparent pathological conditions. Gowers once said that there are states in nerve cells that neither the microscope nor chemistry can detect, but which cause these cells to act very differently to normal cells and hence the sudden discharges. In many cases no changes in the nervous system can be found; but there are conditions that do not reveal themselves to chemistry or the microscope. No one doubts that there are departures from the normal standard. This becomes apparent by the physical and mental changes occurring later on. What was once invisible becomes visible. The normal individual possesses a nervous system that reacts in a standard way to stimuli. The epileptic possesses a nervous system that reacts abnormally to stimuli, whether these come from without the body, or arise within, whether reflex in origin, or due to a central nervous explosion caused by some unknown biochemical process.

From this it must appear that the epileptic of excellent physique and brilliant intellect is far from normal. This is bound to express itself in a departure from the standard of morbidity and mortality. To this it is necessary to direct attention.

The duration of life of epileptics is much below that of non-epileptics. Spratling gives the mean duration at death as 29.4

years, Snell at 33, the Massachusetts Epileptic Institution found it to be 39, and the London County Asylums 40. Dr. W. T. Shanahan, of the Craig Colony, states that a series of nearly nine thousand gave a mean age of 36. Turning to the statistics of the registration areas of the United States for the years 1914, 1915, 1916, and 1917, it is found that there were recorded 10,756 deaths from epilepsy. These deaths are given in quinquennial periods. By a calculation the total years of exposure were obtained. From this data it was determined that the mean average age at death was 35.1 years.

The age of onset is also important. In 1000 cases, tabulated by W. A. Turner, the average age for the first seizure was 15.6 years. In 2383 cases recorded by Gowers, the mean age was 15.4. Dr. Shanahan states that the average onset age of epilepsy approximates 12 years. Dr. Williams, medical superintendent of the Ontario Hospital for Epileptics, gives the average of commencement at about 13 years, and the average age at death as about 34 years. My own cases corroborate these latter figures very closely. It would appear from these statistics that the duration of the disease averages from 20 to 25 years.

The average age at death in the registration areas of the United States from all causes was 42 years. This gives an average of about 8 years longer life than in the case of the epileptics.

It is interesting to compare the epileptic death rate per 1000 with the general death rate per 1000. In the registration areas of the United States it stood thus: In 1914, 19.3 to 13.6; in 1915, 19.1 to 13.5; in 1916, 20.1 to 14.1; and in 1917, 20.4 to 14.2. These death rates for epileptics were found by assuming that there was one epileptic in every 500 of the population, and apportioning per 1000 the actual deaths from the disease.

I shall now submit a table which I prepared from the data in the report of the Craig Colony for the year 1921.

In working out the foregoing table for the year 1896, I took the mean between 0 and 133 for the number throughout the year exposed to death, and for the other years the mean be-

Year	Deaths	No. at End of Year	Death Rate Per 1,000
1896	1	133	14.9
1897	8	214	42.2
1898	14	322	52.2
1899	8	378	22.8
1900	28	612	56.5
1901	36	743	53.1
1902	33	826	42.0
1903	48	831	70.0
1904	47	898	54.3
1905	63	1050	64.6
1906	58	1053	55.1
1907	73	1081	68.4
1908	67	1232	57.9
1909	109	1301	86.1
1910	101	1351	76.1
1911	119	1420	85.9
1912	136	1418	93.7
1913	141	1427	99.1
1914	126	1421	88.4
1915	94	1477	64.8
1916	96	1466	65.2
1917	174	1466	118.7
1918	97	1448	66.5
1919	281	1348	201.0
1920	120	1403	87.2
1921	100	1508	68.7

tween the numbers at the beginning and end of each year. The death rate per 1000 will be seen to be very irregular; but for the ages, very high. It should be noted that severe cases are committed to the Colony.

The manner of death merits attention. Dr. Williams, already cited, states that epileptics are very prone to pneumonia and tuberculosis. He also states that 4 per cent. die of accidents, and 20 per cent. die from some form of nervous disorder. The report of the Craig Colony for Epileptics for 1921 furnishes the following causes of death among 100 deaths. Broncho-pneumonia 15; ileo-colitis 13; lobar pneumonia 13; cardio-vascular 12; epileptic seizures 11; pulmonary tuberculosis 10; exhaustion following seizures 7; cerebral hemorrhage 3; chronic nephritis, septicæmia, pulmonary edema, tubercular peritonitis, and intestinal gangrene, each claimed 2;

meningitis, accident, acute nephritis, inanition, erysipelas, and uterine fibroid were responsible for one death each. Dr. Shanahan claims that 25 per cent. die directly as the result of the disease.

The causes of death are given by Spratling as follows:

Suddenly as result of a fit.....	5 per cent.
From Status Epilepticus.....	23 per cent.
From accidents during a fit.....	12 per cent.
From pulmonary diseases, mainly T. B..	24 per cent.
From organic heart disease.....	10 per cent.
From all other cause	26 per cent.

Seven per cent. of the general death rate is due to external and accidental means.

Two conditions in the United States cause practically the same number of deaths. For the year 1917 there were 3161 deaths among epileptics, and 3044 due to hernia. The following table however points out the marked differences as to ages at death and the duration of life.

Age	Epilepsy	Hernia	Age	Epilepsy	Hernia
Under 5	214	248	55-59	163	319
5-9	117	15	60-64	146	363
10-14	159	8	65-69	114	324
15-19	296	20	70-74	85	307
20-24	299	46	75-79	56	251
25-29	285	69	80-84	24	151
30-34	277	64	85-89	19	75
35-39	308	108	90-94	1	33
40-44	235	138	94-95	0	3
45-49	202	221	100 X	0	2
50-54	143	274	Unknown	10	5

The average age at death of the epileptics, as stated, was 35.1 years, while that of the deaths from hernia was 54. The death rate per 100,000 in both cases over a number of years was 4.

The Scottish Equitable Life accepted twenty-three appli-

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cants during fourteen years with a personal history of some sort of fits. One of these died of tumor of the brain and one of uræmia. Of the remaining twenty-one, the oldest was 65 and the youngest 35, and the average 47. The group, with the extras changed, had been quite profitable. This investigation was made seven years after the last of these had been accepted. From this it will appear that these persons had been on the books of the Company for periods varying from 21 to 7 years. From amongst this class it is possible, therefore, to make safe selections.

Coming to the replies to my circular letter, the outstanding feature in all was that epileptics were undesirable as policyholders. Of the sixty-three answers received, forty-nine or 77 per cent. stated that no epileptics were knowingly accepted on any plan or with any extra rating. Fourteen replied that a few had been accepted. One reply contained these words: "With the exception of an applicant who gives a history of having had epileptic attacks as a child prior to the age of ten, without any seizures over a period of twenty years or more, we have as stated, considered the class uninsurable."

Another reply reads thus: "We might accept a favorable case where the applicant gives a history of having had mild epilepsy ten or more years ago and where he has made a perfect recovery with no effects of any mental or nervous condition during the ten years preceding the application. A case of Jacksonian epilepsy, where a successful operation has been performed and after five years from date of same, might be accepted."

Another reply states that "a few of the mild type have been accepted for small amounts, and on short endowments. No grand mal cases accepted. All cases are rated up or charged extras according to the age, character and cause of the disease. Older ages not accepted. No permanent disability allowed."

In another reply this remark is made: "We have had very few death claims from epilepsy, but even the few that have accidentally been granted policies on incomplete information have given a high mortality rate. In favorable cases we might

accept epileptics of toxic, functional, or traumatic origin on short endowments."

One reply laid down this as its practice: "We would not accept an applicant under 25 who had a history of convulsions possibly or probably epileptic in childhood, but after age 25 we would be inclined to disregard such a history."

The following has been given by another Company: "The applicant must have been clear of the disease for at least ten years before application is made. Very little distinction has been made between the various classes of the disease, such as traumatic, functional, petit mal, etc. Only endowments have been given, and these usually of short period—ten, fifteen, or twenty years. Some few have been clear on endowment plan; others, with a rating or lien. Total and permanent disability has not been granted, and the amount of insurance has been limited."

From another answer the following summary is taken. Grand mal not usually accepted; petit mal cases rated up; with regard to Jacksonian cases, if the cause has been removed and some time elapsed since last attack, they might be considered; only endowments granted; severe types not accepted; in petit mal cases without symptoms for 5 years the rating is 185% and upwards. Epilepsy in infancy and no fits since, calls for but little attention. Total and permanent disability not allowed; the amount of insurance considerably limited; one attack some years ago and cause satisfactorily explained does not necessarily make the case substandard.

The following is the practice of another company. "If only one attack, over ten years previous, no recurrence, we would ignore, giving anything but term insurance. Two cases or more of epilepsy in brothers or sisters would influence our action very much, causing to grant 15 or 20 year endowment, according to age of applicant, with the intention of getting off books at 45 years. Disability is not granted, and the amount of insurance very much reduced."

If one turns to works on Life Insurance but little help is found. Almost invariably the advice is given that epileptics

should be declined. In Chisholm and Pollock's book the following rule is found. "Epilepsy early in life and mild in form as petit mal or an occasional grand mal attack, if some years have elapsed, and applicant 30 or over, may be allowed with a small extra. More severe cases should be rejected unless 15 years have passed since last seizure, and then only with considerable extra."

It will be admitted by all that every class or group can be granted insurance if the rate of mortality for the different ages is known. If a mortality table for epileptics were constructed they could be granted insurance, just as companies now do in the tropics on a tropical rate. There would be epileptic commutation columns, as there are now commutation columns deduced from persons who have become permanently disabled. I hope to have such a table for epileptics before long.

Permit me to take you back to the mortality experience in the registration areas of the United States for 1917, and covering 75,307,906 lives. By a study of the statistics therein contained it is discovered that 50 per cent. of the deaths due to epilepsy had occurred by the end of the 28th year of age; but that, in the case of deaths from all causes, 50 per cent. did not occur until the end of the 44th year of age. These ages apply only to those dying. It will also be discovered from the same statistics that 23 per cent. of the deaths from all causes occurred at ages of 70 and over, whereas in the case of epileptic deaths only 6 per cent. were of ages of 70 and upwards.

From the returns for the United States registration areas for the years 1914, 1915, 1916 and 1917, the general death rate will be found to average 13.8 per 1000. In the case of the epileptic deaths the average for the same four years is 19.9. This reveals the fact that the epileptic death rate is 45 per cent. greater than that of the general death rate from all causes. In estimating the epileptic death rate, one case was assumed in every five hundred of the population in the registration areas.

In estimating the future of epileptics one must remember that the period from 15 to 45 is the one in which the heavy mortality occurs. In the population as a whole this is the

period of low mortality. Put in the form of percentages it turns out that 53 per cent. of the deaths from epilepsy falls into this period, while only 23 per cent. of the deaths from all causes does so.

The best thought of to-day may be summarized as follows:

1. Those who become confirmed epileptics and show the more profound degrees of mental impairment have frequently a family history of the disease or of insanity, but cases with a distinct hereditary strain do much better under treatment than might be expected. If young epileptics reveal a retardation in development of three years, they do very badly.

2. The age of onset is very important. Cases commencing under ten are very unsatisfactory. Most of these cases become confirmed epileptics with marked mental deterioration. Cases coming on between 15 and 25 do much better, the chance of arrest being at least 50 per cent., and not more than half suffering any mental derangement. According to Spratling and Turner, cases commencing between 25 and 35 are usually obstinate, with a large number becoming confirmed. For cases originating from 35 to 45 the outlook is much brighter. Those beginning at the climacteric and in men after mid life almost invariably resist treatment.

3. The duration of the malady is of much moment. Cases that come early under treatment do much better than those who had no treatment for a number of years. The longer untreated the more the mental impairment. Early treatment reduces the frequency of the seizures and saves the mind. If the disease has lasted five or more years without treatment the prospects of arresting the disease are not good.

4. Combination of types should receive attention. The grand mal type yields best to treatment, next the petit mal, and least the combination of these types. In the major type the mental capacity remains in half the cases. In the other half the mind may be badly shattered according to the frequency of the attacks. Petit mal gives rise frequently to mental deficiency of a mild type. The combination of both forms does most damage to the mental faculties.

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5. The more frequent the seizures the less likelihood of an arrest of the disease. It is recognized, however, that cases that have a regular monthly seizure do not do as well as those who have attacks more frequently at irregular periods. Cases that have fits in series with intermission develop dementia very frequently.

6. The longer the intervals the better, whether the result of treatment or a spontaneous condition. No case should be considered as cured unless eight to ten years have elapsed without a fit. About 12 per cent. can be cured.

7. Cases that arise from some organic disease affecting the cortex, unless such can be surgically dealt with, yield a gloomy prognosis.

With regard to insurance two problems must be considered. The first is that of applicants with a family history of the disease, but who have not themselves suffered from it. It will be remembered that at least 80 per cent. of the cases commence by the age of 20. If the applicant is 25 and in good health the risk from epilepsy is almost negligible; and a very slight extra would afford protection to the Company. Gowers states for a person with a hereditary tendency but in good health at 20, his chance is as 1 to 50 that the disease will not develop.

The second problem, namely the issuing of policies to those who have developed the disease gives rise in turn to several other problems.

1. Applicants who give a history of slight epileptiform attacks, not often repeated, and that may have been due to some derangement of digestion, exhaustion, over nervous strain, or temporary weakness of the heart, but who at time of examination are in good health and with freedom from epilepsy or insanity in the family may be accepted on any plan with a rating to cover 135 per cent. mortality, or from 7 years at age 20 to 4 years at 45.

2. Those who have been free from seizures for 8 or 10 years and are in good health, may be accepted on any endowment plan up to 30 years with a rating to cover 150 per cent. or from 13 years at age 20 to 6 years at 45.

3. Those who have fewer than four seizures yearly and retain a good physical and mental condition, may be granted endowment insurance up to 20 years duration with a rating to cover 175 per cent. or 19 years for age 20 to 9 years for 45.

4. Cases with slight mental impairment but physically robust, might be granted endowments up to 15 years duration with a rating to cover 200 per cent. mortality, which would run from 25 years at age 15 to 11 years at age 45.

5. All cases with a combination of mental and physical stigmata should be refused; also when the seizures are four a year, or serial, or status in type.

6. All cases, no matter what the type, should be declined if the insurance is taken out by some second party in a speculative way.

7. All cured cases and cases of a favorable nature who are wage earners, and insuring on their own account by an investment policy, or for the protection of dependents, are thereby entitled to the most lenient consideration, the group to which they belong will permit.

8. When it is borne in mind that the duration of the persistent disease from inception to death is but little over 20 years as an average, the date of commencement of the seizures becomes very important in determining the kind of policy that should be granted. Examiners should be specially diligent in the efforts to elicit this information.

9. Liens do not suit this group of risks. The risk is an increasing one in the large majority of cases, and therefore demands a constant extra to the premium, such as obtained by adding on a number of years to the present age.

10. The age of onset is important, as already pointed out. Organic cases unrelieved are bad and the duration of the malady untreated bears on the prognosis.

My paper is perhaps too lengthy and liberty is granted to cut it down for the published transactions. I have gone into the subject exhaustively as it has never been taken up and I desired to tell the truth, the whole truth and nothing but the truth about the epileptic class. Companies have now prac-

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tically agreed to ignore hernia except in bad cases which causes a death rate of 4 per 100,000, while they are very severe upon epileptics who cause the same death rate. The only difference being that the deaths among the former occur at the average age of 54 whereas in the latter it is 35. This, however, is a matter of adjustment, as is done in so many other substandard groups. If epilepsy was of one type the problem of determining what to do would be comparatively easy, but it is a disease of many types and multiple exciting etiologies, and herein lies the difficulty.

In the chaff of my paper there may be some grain, on its pages some fruit, in the midst of its verbiage some ideas.

Dr. McMahon—Dr. Jenney, one of our newly elected members, and Secretary of the Medical Section of the American Life Convention, will lead in the discussion of this paper on Epilepsy.

Dr. F. L. B. JENNEY: When I received a request from Dr. McMahon to discuss this paper I wrote him that I did not feel that I was fitted to do so, as I had had no experience in the selection of epileptics and had never given much thought or study to Epilepsy. I wrote the doctor that there must be in the Association some one who was far better fitted than I to undertake the discussion of this paper but that I would do what I could if he still felt I was competent. He replied that he was anxious to secure as wide an expression of opinion as possible upon this subject and again urged me to prepare a discussion, which will be my excuse for appearing before you.

Before receiving a copy of Dr. Ferguson's paper I firmly believed there was not only little, if anything, to be said on the subject, but also that a company would not knowingly accept an epileptic or an applicant who gave a history of having been a victim of that disease, unless, perhaps, in a few cases of mild Epilepsy where a number of years had elapsed since the date of the last attack.

I was distinctly surprised on reading this excellent paper to

learn that there was so much to be said in favor of these unfortunates. At this time I want to congratulate Dr. Ferguson on the excellence of his paper, and commend him for the exhaustive study he has made of the subject, and for the clear manner in which he has presented his array of facts.

It is fortunate for insurance companies that Epilepsy is essentially a young people's disease, and that about 50 per cent. of all cases show a neurotic inheritance, as Dr. Ferguson has shown. Most of our applicants for insurance apply after 20 or 25 years of age, and 80 per cent. of cases commence before age 20, and 85 percent. before age 25, so careful examinations and inspections would eliminate the great majority of epileptics.

In 50 per cent. of all cases can be found a direct or indirect neurotic history, as shown by Dr. Ferguson, and care should be exercised in cases showing a family history of insanity or Epilepsy to be sure that our applicants are mentally sound, as Epilepsy and insanity are interchangeable in families. We should also select with care when it is known that either parent has had Syphilis.

In estimating the future of epileptics, Dr. Ferguson has advanced rules summarizing the best thought of today. Therein he shows that in cases where the age of onset is between 15 and 25 years the chance of arrest is about 50 per cent. Where the onset is under 10 they are unsatisfactory; where the onset is between 15 and 25 they are fairly satisfactory; where the onset is between 25 and 35 they are usually obstinate, with a large number becoming confirmed. Where the onset is between 35 and 45 the outlook is much brighter. Those beginning at the climacteric and, in men, after middle life almost invariably resist treatment.

From the above it would appear that the most satisfactory cases would be those in which the onset of the seizures is between 15 and 25 years of age, or between 35 and 45 years. The doctor shows that combination of types should receive attention. The Grand Mal yields best to treatment, next the Petit Mal and least the combination of these types, these latter showing the most damage to the mental faculties.

The frequency of the seizures is also important and cases that have a *regular* monthly seizure do not do as well as those that have attacks more frequently at *irregular* periods. No case should be considered as cured unless 8 or 10 years have elapsed without a seizure.

It seems to me that as Medical Directors we would have considerable difficulty in obtaining from our examiners reliable information regarding our cases, such as the age at onset of the seizures; whether the attacks are those of Grand Mal, Petit Mal, or a combination of the two; whether the attacks occur regularly or at irregular periods; the frequency and severity of the seizures, etc. In some cases the applicants, even if wholly honest and truthful, would be unable to give this information. I have known of cases in women that were found, after marriage, to have Nocturnal Epilepsy, but the occurrences were unknown to them and had probably existed for several years or from childhood, and in those cases no examiner could have given the age at onset or the character or frequency of the seizures before marriage. In cases that had been treated by competent neurologists who had taken careful histories, we might get the information we desire, but I doubt if we could get it in the majority of cases.

In the selection and rating of applicants who give a history of this disease, Dr. Ferguson has given us rules for our guidance. He begins with those who give a history of slight epileptiform attacks, not often repeated and probably due to some derangement of digestion, exhaustion, over nervous strain, etc., but who at the time of examination are in good health and with freedom from Epilepsy or insanity in the family; and he goes on down through cases that have been free from seizures for 8 or 10 years; those who have fewer than 4 seizures a year; those who have slight mental impairment but are physically robust and those with a combination of mental and physical stigmata. On the first of these he would impose a rating of 135 per cent. on any form of insurance; to the second group he would issue endowments rated 150 per cent.; to the third, endowments rated 175 per cent.,

and to the fourth he would issue endowments rated 200 per cent.

Again we are confronted with the difficulty of gathering the proper data to enable us to fix the mortality rating by definite classification of our applicants.

The doctor would decline all cases with a combination of mental and physical stigmata; also those in which the seizures are 4 a year or more; are serial or status in type. He shows that the duration of the disease from inception to death is but little over 20 years on an average, hence the date of the commencement of the disease becomes very important in determining the kind of policy that should be granted, and he suggests that examiners should be especially diligent to elicit this information.

He suggests that, as the hazard is an increasing one, liens are not suitable to this group, demanding rather, a constant extra to the premium by advancing the age of the applicant.

Personally, I would not care to accept any applicant in any of the groups outlined by the essayist, except those in groups one and two before mentioned. Those having actual epileptic seizures, even if fewer than 4 per year; those with slight mental impairments, and those with a combination of mental and physical stigmata, I would much prefer to let alone until such time as *insurance* statistics have been compiled covering a sufficient number of cases and covering a period of years.

There is considerable doubt in my mind as to the relative value of statistics derived from the experience of epileptic colonies presided over by experts, and those of life insurance companies compiled from histories obtained by the rank and file of examining physicians. As already pointed out, the latter would be, in my opinion, much more unfavorable than the former, because of improper classification of the cases, insufficient information as to the date of onset of the seizures, etc., and also because of the ever present selection against the companies.

In closing, there are two points that I would like to have Dr. Ferguson explain more fully.

He states that the Grand Mal cases yield best to treatment, better than the cases of Petit Mal, yet he gives his most favorable rating to those cases of slight *epileptiform* attacks, not often repeated, and due to some derangement of digestion, exhaustion, overwork, etc. This class seems to me to include many cases of Petit Mal as well as cases that are not true Epilepsy.

I would like to have explained, if possible, why it is that the best results of treatment occur in cases where the onset of seizures is between the ages of 15 and 25, and the next best results in cases beginning between ages 35 and 45, and why the cases beginning between ages 25 and 35 "are usually obstinate." What extra ratings, if any, would be imposed upon those applicants whose seizures begin between the ages of 25 and 35?

Dr. McMahon—I am sure you will all agree with the wisdom of my selection in asking Dr. Jenney to discuss this paper. We will now hear from Dr. English.

Dr. C. H. ENGLISH—*Mr. President and Gentlemen*—Dr. Ferguson has so concisely and comprehensively presented to us the subject of epilepsy in all of its phases that it seems to me there is very little further to suggest. I believe that I voice the sentiment of this body in saying that from a life insurance and under-writing standpoint we shall ever be under obligation to Dr. Ferguson for this masterful correlation of facts.

A few words with reference to the features of this paper which stand out as guide posts or land marks, as it were, for us to follow in our future work in dealing with this class, would seem appropriate.

Referring to the number of epileptics in the registration area of the United States and Canada, it is safe to believe that we have at least 250,000 epileptics and, I think it is also safe to say, that we have at least an equal number who are dependent upon them for support, making a total of 500,000 of our combined population that is entitled to insurance consideration just as much as those who are standard life insurance risks, if just and adequate ratings can be determined.

The classification based upon aetiology is certainly one of the most valuable features of this paper and, as suggested by Dr. Ferguson, its careful consideration by every Examiner in dealing with cases of epilepsy will assist us very greatly in arriving at a more comprehensive classification. I think all will agree that it is most difficult to justly classify epileptics. It is very difficult to obtain full information as epileptics are extremely secretive, reticent and inclined to conceal any information that would help us to properly classify them. If this classification or something similar were forwarded to the Examiner, it would assist him very greatly to clarify the haze in his own mind and give us some tangible facts upon which to classify in the Home Office.

The morbidity, mortality and also the causes of death are very fully covered in this paper. The table comparing death from epilepsy with death from hernia, at the different ages, is very interesting. A large number of deaths from epilepsy occur in early life, while the reverse is true of hernia, which occur in old age when degenerative processes have set in; therefore, the deduction that in epilepsy the heavy mortality is experienced between the ages of 15 and 45, while in the population as a whole this is the period of low mortality.

In the summary of the best information we have to date, we find the following in paragraph 2:

"The age of onset is very important. Cases commencing under 10 are very unsatisfactory. Most of these cases become confirmed epileptics with marked mental deterioration. Cases coming on between 15 and 25 do much better, the chance of arrest being at least 50 per cent. and not more than half suffering any mental derangement. Cases commencing between 25 and 35 are usually obstinate with a large number becoming confirmed. For cases originating from 35 to 45 the outlook is much brighter. Those beginning at the climacteric and in men after mid life almost invariably resist treatment."

You will note that "For cases originating from 35 to 45 the outlook is much brighter" is followed by "Those beginning at the climacteric and in men after mid life almost invariably

resist treatment." I am inclined to believe that the thought carried in reference to those between 35 and 45 is not what Dr. Ferguson would have us believe; at least, it does not seem to me that the foregoing and following statements harmonize and, thinking it possible that this is a typographical error, I shall be pleased to have Dr. Ferguson explain his intended thought in his closing remarks.

In paragraph 6 of this summary, it is stated that "No case should be considered as cured unless eight to ten years have elapsed without a fit. About 12 per cent. can be cured." This basis as to time would seem to me rational; however, many are inclined to the opinion that those showing a shorter time without a fit should be taken as standard.

With regard to the acceptance of these cases, giving either a family or personal history of the disease, would state that the classification as suggested by Dr. Ferguson fully coincides with my own opinion and I am led to believe that if these applicants are accepted under these three classifications, the companies accepting them will be adequately safeguarded and a large number of people will be given protection at equitable rates.

The Company with which I am associated has to date accepted about 100 cases with a family or personal history of epilepsy at approximately the same classification and rating as that recommended by Dr. Ferguson. The period of exposure, however, is slightly less than three years on the group, so that our experience, based upon this small number and short exposure, is absolutely valueless. To date we have had no deaths in this class.

It is very encouraging indeed to know that other companies are accepting these risks and may I express the hope that a greater number of companies will launch out in this field, so that in time we shall have just as valuable a basis of underwriting principles in regard to the acceptance of epileptics as we now have for many other impairments.

Dr. Fisher—Mr. President: Along the lines of this paper on Epilepsy, the Northwestern Mutual accepted 505 cases

during the years 1885 to 1908, carried up to 1915, where one parent was living or died of epilepsy, and the mortality was about two-thirds of the expected.

Dr. Mahon—Thank you, Dr. Fisher, that is a most interesting contribution. We will now have presented Dr. Beckett's paper—"Tuberculosis in its Relation to Life Insurance."

TUBERCULOSIS IN ITS RELATION TO LIFE INSURANCE

By

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From an insurance standpoint probably no other disease so much concerns the Medical Director as does tuberculosis.

Tuberculosis has been called the great white plague, a disease of the masses, on account of its great prevalence among all classes of people. It is not confined to climates or races. It is universal. For hundreds of years it has been the most prevalent, most feared, and probably the most fatal of all diseases. Pulmonary tuberculosis was described as far back as 460 B.C. as the most difficult disease to treat, and fatal to the greatest number. In 1550 a celebrated physician declared consumption to be one of the most dangerously contagious and most easily contracted of diseases. In 1682-1771 the celebrated anatomist, Morgagni, would never perform an autopsy on an individual who had died from tuberculosis, for fear of infection. In 1782 Naples, by a royal decree, ordered the isolation of consumptives, and a disinfection of their personal effects, furniture, etc., by means of vinegar, brandy, lemon juice, sea water, or fumigation. Any violation of this law was punished, if the individual was an ordinary mortal, with three years in the galleys, and if he happened to be a nobleman he was sent for the same time to the fortress, and had to pay three hundred ducats. The physician who failed to

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notify the authorities of the existence of a tuberculosis patient was fined three hundred ducats for the first offense, and a repetition of the neglect would banish him from the country for ten years. There was a law in Spain and Portugal during this time which obliged the parents or nearest relative of a consumptive to notify the authorities when the patient had arrived at the last period of the disease. This was done for the purpose of making sure of the disinfection of the personal effects of the patient after his death.

In the first half of the 19th century little attention was paid to the infectious theories of tuberculosis, even by medical men. The contagiousness of the disease could not be scientifically demonstrated and although there were physicians here and there who believed in the infection of the disease nothing positive was taught regarding it at the centers of medical learning.

In about 1850 the prevalence of tuberculosis began to decline, and has been declining ever since. The first 20 years following the establishment of the Volunteer Tuberculosis Association, tuberculous mortality decreased from 238 per thousand of population to 164 per thousand, a decrease of 30.25 per cent. A comparison of five year periods during the past 15 years show a tuberculous decrease of 22.2 per cent. Not only has improved treatment and management done much toward lessening the fatal results of tuberculosis, but improved sanitation and living conditions have been a material factor in bringing about this result. In any large city you will see fewer tuberculous people on the street to-day than you would have seen 25 or 30 years ago. If you would ask anyone how many among their friends or relatives are now afflicted with tuberculosis, the general answer would be none; while only a few years ago almost everyone had some relative or acquaintance who was ill with that disease. More tuberculous patients to-day recover than probably at any prior date in history. The disease is better understood and better treated. The laity better understand the precautions that should be taken to prevent infection, and also better understand the

management of the disease necessary to affect a cure. More cases are recognized in the early stages, when an arrestment or cure can more easily be effected. With proper treatment and care during the early stages from 80 to 90 per cent. of recoveries may be expected, with from 10 to 20 per cent. of failures; while in the advanced stages these figures may be reversed, that is from 80 to 90 per cent. we should expect less favorable results. Dr. Pottenger in a recent excellent article on the present status of tuberculosis says that: "There are two methods of approach to the cure of an infectious disease; one by specific remedies which combat the organisms or their toxins directly, such as anti-toxin in diphtheria and quinine in malaria; and the other by aiding the natural defensive forces of the patient so that he cures himself. So far, tuberculosis has baffled search for a truly specific remedy; and while the search for such a measure still goes on and the hope of some such boon to the race being discovered is deeply imbedded in the minds of investigators, yet many of those who are most interested in the solution of the tuberculosis problem have greater hope that some method of vaccination may be discovered which will protect the individual against massive infection, during the early years of life." It is not only important that early treatment should be instituted, but it should also be carried out long enough to fully cure the patient. Most early cases are not treated for a sufficient length of time, and on that account relapses or re-infections are more apt to occur. Tuberculous infection is more common during the early ages, and probably more deaths from tuberculosis occur between 25 and 35 years of age than during any other like period.

The National, State and Municipal governments are doing much to improve sanitation and prevent epidemic and contagious disease, as well as to care for the indigent tuberculous population. The National Tuberculosis Association has done a wonderful work, and it would be impossible to estimate the good that will come from the efforts of that Association. The Metropolitan Life Insurance Company of New York is doing

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our country a great service. The Framingham demonstration is the most conspicuous illustration. We should have a National Health Department presided over by a Cabinet Secretary; this is surely as important from a health and economic standpoint as is a National Agricultural Department presided over by a Cabinet Secretary.

In all the states the mortality from tuberculosis has very materially decreased in the last few years. There does not seem to be any special locality in our country where this disease prevails to a very much greater extent than another. Kansas seems to have the lowest mortality from tuberculosis, while Colorado, New Mexico, Arizona and California have a high mortality rate. This undoubtedly is due to the indigent migratory tuberculous population in these states, as well as a large Indian and Mexican population, which are especially susceptible to this disease. Colorado, New Mexico and Arizona are considered our Health Resort States, and a very considerable percentage of the population have tuberculosis or have come to these states because of some member of their family who was ill with this disease.

Just how far heredity plays an important part in transmitting a susceptible tendency to tuberculosis cannot well be determined. We inherit our build, the shape of the head, the color of our eyes and hair, certain traits of character, certain mental tendencies, and we surely inherit a physical condition which pre-disposes to certain diseases. Probably the most frequent of these diseases are tuberculosis, apoplexy and insanity. There also seems to be an heredity family trait as to the age at which death from tuberculosis occurs. While in most families the age is between 25 and 35 years, in other families it may be between 35 and 45 years. The farther an individual lives beyond the family trait age the more favorable is his chance for escaping the disease altogether. I believe it is a well established fact that any individual who has a tuberculous family history as a rule is more susceptible to the development of that disease at any age than is an individual who has no such family history. Applicants who have a per-

sonal or family tuberculous history, or have through inheritance a constitution that is below par, die early. It matters little whether they die of tuberculosis or some other disease—they die, and the Company pays the claim.

It has been difficult to get any statistics that are of value on the ultimate results of apparently cured tuberculous patients. I have communicated with several Sanatoria and County and State Health Departments regarding the end results of their cured patients, but have failed to secure data that would be of much importance. The patient who has a recurrence of tuberculosis may not return to the sanatorium or to the doctor who treated the case, and no subsequent record may be available. These relapses usually occur during the first five years after the patient has been discharged.

From one of our leading specialists (Dr. C. C. Browning), I obtained a record of over three thousand tuberculous patients. His records showed that the percentage of relapses or re-infection, after the patient was pronounced as cured or arrested, to be 40 per cent. This coincides with the experience of the Pacific Mutual Life Insurance Company in claims for death or permanent total disability in applicants giving a previous history of tuberculosis. In other words, about two out of every five applicants giving a previous history of tuberculosis who were pronounced cured, became either disability or death claims within the first five policy years. Applicants who were treated in well established sanatoriums and discharged as cured and who upon examination were found to be in good physical condition and pursuing healthful occupations showed much better resistance than those who were privately treated. Applicants with a tuberculous history showed poor resistance during the influenza epidemic, or after attacks of pneumonia, and the mortality in these groups was very high.

In 3045 patients examined, 1351 (44%) gave a history of previous attacks.

1299	gave a history of	pleurisy
1211	" " " "	bronchitis
1064	" " " "	asthma

755	gave a history of influenza
594	" " " " pneumonia
743	" " " " frequent colds
337	" " " " shortness of breath
709	" " " " typhoid
1335	" " " " general appearance—good.

In another group of 2653, where the family history was recorded, 1420 gave negative family histories, and 1233 showed a tuberculous family history. In a group of 2909, 1155 gave a history of exposure to tuberculosis, and 1754 did not. In a group of 3544, 1841 live in the city, 1037 live in villages, 666 live in the country and out-of-door life. In a group of 3435 cases, 1692 were male, 1743 were female. Condition of health prior to illness, in a group of 2425 cases, 1506 good, 755 fair, 164 poor. In a group of 2466 cases, 1957 were engaged in an active occupation, and 509 lead a sedentary life.

Deaths from tuberculosis, all forms, in California—1921. Total, 5427. Male, 3465. Female, 1962.

Race	Population	Deaths
White	3,300,000	4905
Negro	39,000	148
Indian	17,500	50
Chinese	29,000	170
Japanese	72,000	146
Other		8

Residence in California	Nativity
Under 1 Mo.	69
1 to 3 Mo.	169
4 to 6 Mo.	200
7 to 12 Mo.	395
1 to 4 years	859
5 to 9 years	635
10 yrs. and over	2554
Unknown	546

California	1315
Other U. S.	2101
Foreign	1932
Unknown	79

Of the 1654 tuberculous patients discharged from the sanatorium for from four to nine years Rutledge and Crouch found 771 or 46.6 per cent. are living and 883 or 53.4 per cent. are

dead. In 425 or 25.7 per cent. tubercle bacilli were not demonstrated in the sputum; of these 357 or 86.4 per cent. are living and 68 or 13.6 per cent. have died. Thirty-five of the 68 patients who died under this classification, died from causes other than tuberculosis. Eighty-nine were classed as being in the incipient stage with a positive history of tuberculosis; 76 of this number or 85.4 per cent. are living and 13 or 14.6 per cent. have died. Of 96 moderately advanced cases 87.5 per cent. of the patients are living and 12 or 12½ per cent. are dead. Of the 24 far advanced cases 13 or 54.25 per cent. are living and 11 or 45.75 per cent. have died. There were 119 doubtful cases with 112 or 94.1 per cent. living and 7 or 5.9 per cent. dead. During four years, in 1229 of the 1654, tubercle bacilli were found in the sputum; this was 74.3 per cent. of all cases; 413 or 33.6 per cent. are living and 816 or 64.4 per cent. are dead. Eighty-four were incipient, with 62 living and 22 dead. There were in all 495 moderately advanced cases with bacilli in the sputum, of which 259 or 52.3 per cent. are living and 236 or 47.7 per cent. are dead. Of 654 advanced cases 92 or 14.2 per cent. are living and 558 or 85.2 per cent. are dead.

The Trudeau Sanatorium has succeeded in tracing 814 patients who have been discharged from the institution 20 years or more. Of these 666 or 81.8 per cent. are dead, while 148 or 18.2 per cent. are living.

According to R. F. M. Picken, the expectation of life of male sanatorium cases discharged with the disease "arrested" is estimated at somewhere about 14 years.

Sufficient time has now elapsed since the world war to enable us to determine the effect of poison gas upon the lungs. We would consider those cases that have had no trouble since recovering from the acute condition to be normal cases at the present time.

Climate has always been considered an important factor in the treatment of tuberculous patients. While there is no specific climate for tuberculosis, still a climate that is dry, where almost every day the sun shines, and where the patient can live almost continuously day and night out-of-doors, will

doubtless do more for the relief of this disease than any other climate. Thirty-five years ago Southern California might have been considered an open-air sanatorium for those suffering with tuberculosis. Years ago those suffering from tuberculosis from all parts of the country came to Southern California with the assurance on the part of their physicians and friends that this climate would absolutely cure them. Alas, their hopes were too often blasted. Now, Colorado, New Mexico and Arizona are the favorite states for these patients. Instead of coming to Southern California they are advised by their physicians, if a change of climate be deemed best, to go to one of these states, where there is almost continuous sunshine, and very few rainy days. In the winter the lower parts of the country are warm and the patients are able to sleep out-of-doors, while in the summer when it is hot the patients can go to the hills and mountains where the climate is still pleasant and the sun continues to shine.

There are being established in these states well conducted Sanatoria that are caring for the active cases. Rest doubtless is the first essential in the treatment of active tuberculous cases, and this along with medicinal treatment is best carried out in a sanatorium. I do not believe that climate is especially essential to the treatment of tuberculous patients during the active stage. In other words, I would rather be treated in a poor climate in a first class sanatorium than in a good climate outside a sanatorium.

After there is an arrestment I believe that there are no parts of our country better adapted to the building up and full recovery of the patient than in some portions of Colorado, New Mexico, Arizona and California. These cases should remain in the districts in which they have recovered, as they are more apt to have a recurrence if they return to the more rigorous climates where they have formerly lived. I believe it is also a fact that people are less apt to contract tuberculosis in the districts mentioned above than they are in most other states of our country.

Environment and occupation are important factors in the

development and progress of tuberculosis. Unfortunately there is a large class of our population, who by force of circumstances, cannot obtain the necessary care and treatment which are essential to the cure of this disease. Poverty and tuberculosis go hand in hand. The highest mortality in any large city is found in its poorest sections. Filthy slums and the crowded tenement sections with bad ventilation and insufficient light are prolific nests for the development of this disease. It is difficult to get any definite facts about the relationship of special occupations to tuberculosis. There are so many diverse factors to be considered. Lilian Brandt's summary covers very completely the conditions which may make an occupation favorable for the development of tuberculosis. (1) Low rate of wage entailing poor home conditions. (2) Unsanitary conditions of place of employment. (3) Exposure to dust from marble, stone, plaster, wood, metals or textiles. (4) Excessive physical exertion or constrained position. (5) Close confinement within doors. (6) Exposure to excessive heat. (7) Temptations to intemperance. (8) Long or irregular hours.

Does the arrested pulmonary tuberculous case ever become on a par with a normal individual? Should we not always consider the best of these cases slightly sub-standard? Tuberculosis of the bone, joints or cervical glands is usually local, and if occurring in early life is frequently entirely cured. The insurability of these cases will depend largely upon the extent of the lesion and the time involved in the cure. Those cases that have become chronic especially hip joint and spinal cases in my mind are never insurable on standard plans. These cases seldom live out their expectancy. Cervical adenitis usually occurs in childhood, and if suitable surgical treatment is promptly given there seems to be little affect on the individual's longevity, provided the individual is otherwise normal. Wallgren examining the records of the Upsala Clinic found that of 526 cases of tuberculous glands given operative treatment from 10 to 30 years ago, he was able to trace the history since in 251 cases. Of the 79 with sound lungs at the time of the operation 63 still are free from pul-

monary disease, 9 have pulmonary tuberculosis, 5 had miliary tuberculosis and 2 tuberculous meningitis. Of the total gland cases 16.5 per cent. had pulmonary tuberculosis at the time. Of the total 224 traced cases, 3 per cent. had terminated with meningitis and 4 per cent. in miliary tuberculosis. Among 516 tuberculous persons given treatment at the Chest Clinic at Upsala only 1.7 per cent. had a history of enlarged and probably tuberculous glands before the onset of the pulmonary process. Surgery may do much in the relief of certain local tuberculous conditions, but seldom, if ever, places the individual in that normal state of health that existed before he acquired the tuberculosis. Surgery may relieve tuberculosis of the ovaries and tubes, the peritoneum, retro-peritoneal glands, the plural cavity, but do these cases ever return to a par with the normal individual? In my opinion they do not. Pleurisy following pneumonia or influenza is usually due to a pneumonic or streptococcic infection, and is not tubercular. These cases usually recover promptly and do not become an insurance hazard after a sufficient length of time has elapsed to assure us that they are completely cured. Empyema following these cases as a rule recover promptly, after drainage has been established. These cases become safely insurable if the case is otherwise normal. Such cases as tuberculosis of the kidney, stomach, intestines or mediastinal glands need not be considered. Syphilis as a complication of tuberculosis is a serious impairment, and such cases never become safely insurable at standard rates.

Tuberculosis as a general rule brings about a condition of hypotension, and this hypotension is secondary to the tuberculous process. Thayer's figures are as follows:—

Age	Systolic Pressure
10 to 20	100
20 to 30	110
30 to 40	94
40 to 50	105
50 to 60	105
Over 60	114

I quite agree with Dr. William Bradford Bartlett when he states that it is his belief that no case giving a history of indisputable pulmonary tuberculosis should be considered as insurable at normal rates for ten years after the attack, and even then cases should be selected with very great care.

Tuberculosis of the appendix occasionally occurs with no other recognizable lesion in the gastro-intestinal tract. These cases are usually associated with pulmonary tuberculosis. I have operated several such cases.

Pregnancy is very frequently the starting point of tuberculous disease. As a rule pregnancy aggravates the symptoms of the tuberculous process, which subsequently becomes acute and runs a fatal course. A woman with a tubercular tendency may develop this disease with any difficult confinement.

Probably no other problem in the selection of life insurance risks causes the Medical Director more difficulty than do those risks having a tuberculous personal or family history. There are so many conditions to take into consideration, which really have a bearing on the insurability of the applicant.

Applicants under 25 years of age with a bad tuberculous family history we consider unfavorably unless they are well past the family tubercular age, and are of good weight and good environments. If there be only one case of tuberculosis in the family then we would look upon the case more favorably. Applicants who give a history of having had tuberculosis are postponed until 10 years after they have been pronounced as cured. Severity of the attack, the length of time of treatment, the general health of the applicant, his weight and measurements, his place of residence, his habits of life, and his exposure to the disease are all taken into consideration in passing upon these risks. The Pacific Mutual Life Insurance Company does not do a sub-standard business, and many of these cases that we reject could be taken with an increased rating that would cover the extra hazard.

I realize that the disease of tuberculosis as effecting Life Insurance is a big subject, and that I have in this paper given nothing that is new, but hope that a free discussion will elicit

much that will be of value in deciding many of these difficult cases.

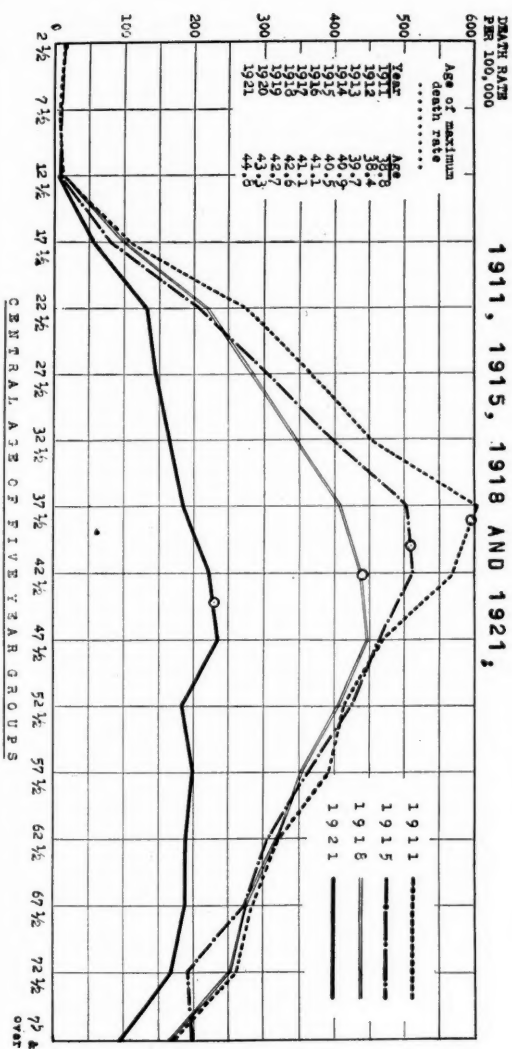
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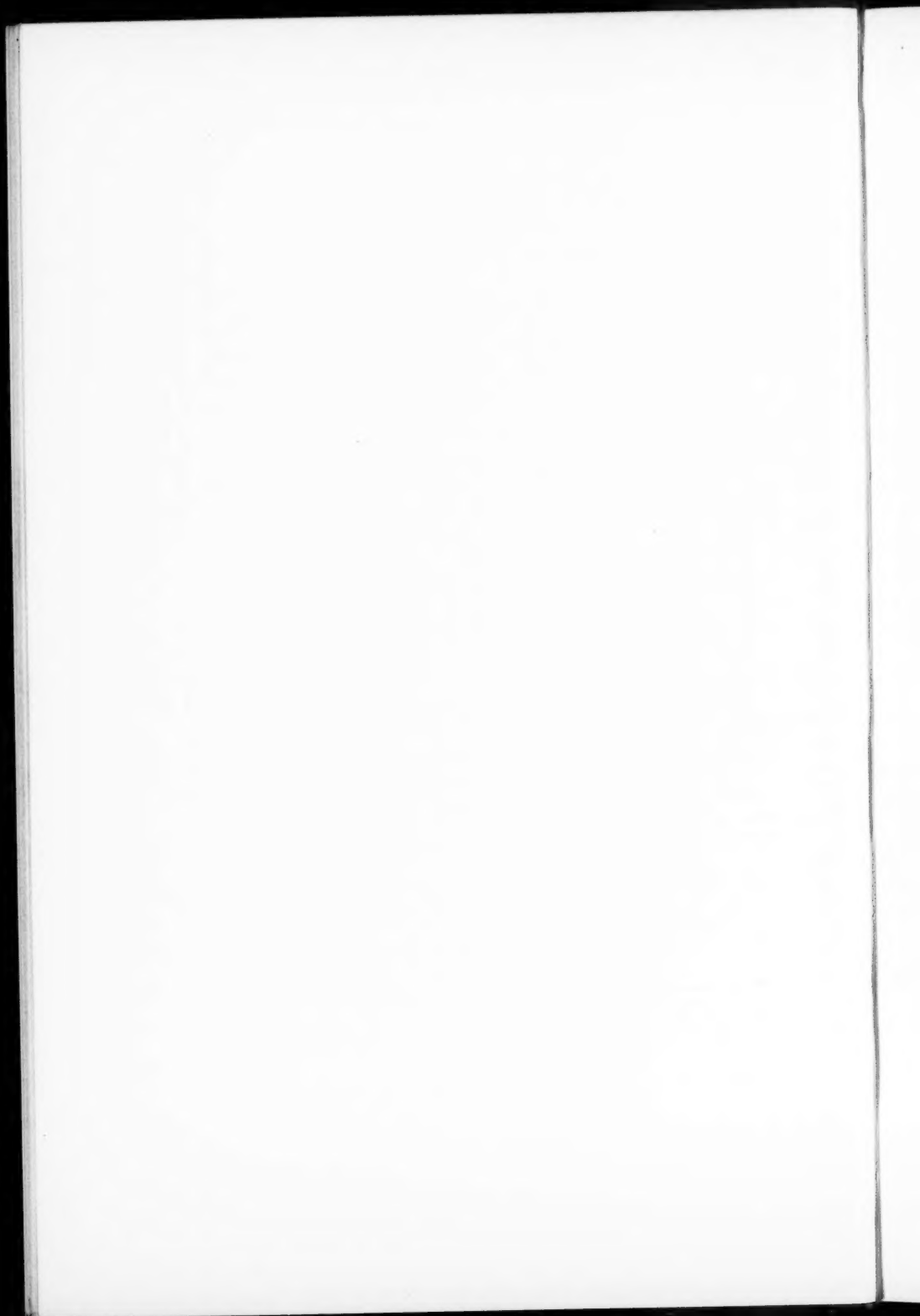
- TICE. *Practice of Medicine. Tuberculosis.* Vol. II. Prize Essay. KNOPF.
History of the National Medical Association. KNOPF.
 ARMSTRONG. *Journal A. M. A.*, vol. lxxii., p. 67.
 ARCHARD. *Bul-de l'Acad. Med.*, 1919. No. 5, p. 135.
 RUTLEDGE & CROUCH. *Ultimate Results of Sanatorium Treatment of Tuberculosis.*
American Review of Tuberculosis, Baltimore, Nov., 1918.
 WALLGREN. *Upsala Lakareforeings Forhandlingar*, July, 1918.
 BALDWIN. *American Review of Tuberculosis*, Baltimore, Aug., 1919.
 MURPHY. *Boston Medical & Surgical Journal*, Aug., 1919.
 PALMER. *General Health Activities and Their Effect on Tuberculous Mortality.* *Journal A. M. A.*, Sept., 1919.
 Trudeau Sanatorium. *American Review of Tuberculosis*, Baltimore, Oct., 1919.
 POTTENGER. *Present Status of Treatment of Tuberculosis.* *Journal A. M. A.*, vol. lxxix, p. 729.
 PICKEN. *Expectation of Life in Pulmonary Tuberculosis.* (London) *Lancet*, 1919, p. 108.

Dr. Knight—Mr. President: I have had no time for preparation since I received your letter asking me to say a word about this paper and I am only going to refer to two or three points. Dr. Beckett says that the death rate from tuberculosis has been going down for the last thirty or forty years and he is right. But I think it has been going down even faster than he says in his paper and that it is still going down. He quotes the gains at about 20% to 22% in the last fifteen years. In our Company's Industrial Insurance experience, the death rate has gone down 50% since 1911 and is still going down. Dr. Dublin has given us a chart here that is quite interesting and illuminating.

In 1911 the death rate from pulmonary tuberculosis was 203 per 100,000 and in 1921 the rate was 105.6. This curve in the chart shows very clearly how that death rate has gone down

DEATH RATES PER 100,000 FROM TUBERCULOSIS OF THE LUNGS
AT SPECIFIED AGE PERIODS. WHITE MALES IN
INDUSTRIAL DEPARTMENT, METROPOLITAN LIFE INSURANCE COMPANY.
1911, 1915, 1918 AND 1921,





for the ages of the working period of life. Practically most of the saving has been of men not only in the working classes but most of it during the working ages. You will see how the average maximum age has gone from 38.8 in 1911 to 44.8 in 1921.

We have proven too that in an intensive campaign against tuberculosis, in a limited population as in Framingham, Massachusetts, the death rate has been brought down 50% (from 120 per 100,000 to 60 per 100,000) in five years.

Dr. Beckett speaks quite a bit about the after life of the sanatoria patients. We have had quite interesting experiences with our employees after treatment at Mount McGregor. About 80% of all the discharged cases, since 1913, when the place was opened, to the end of 1920, are known to be at work. 10% are unable to work, and 10% have died. Of all the incipient cases on admission, 90% are at work and 7% are alive but unable to work, and 3% have died. We have much to learn yet about these after life mortalities of the arrested tuberculous patients. I won't try to go into any figures now, because they are handled splendidly by Dr. Rogers and Mr. Hunter in the paper before you, but I am quite sure that they in their Company have been too liberal in these cases that have been tuberculous, and in fact they say so themselves.

Dr. Beckett raises the question whether an arrested pulmonary case ever becomes on a par with a normal individual. Our experience with these cases is that they were kept as patients at the Sanatorium for long average periods of time until they not only had the disease arrested, which is important, but until they were supposed to be well enough to stay well and to stay at work, which is much more important. Even under those good conditions, the actual death rate, with due regard to their ages and as compared with the general death rate among Metropolitan Life Insurance Company employees, is just 200% of the expected. We get this notwithstanding our reasons for feeling so happy about what we can show you in the Home Office—300 or more arrested cases, all apparently staying well, we examining them from month to

month, weighing them, sending them away if they are not apparently doing well and doing all in our power to help them maintain their good health. Thus you will see that these results are most encouraging but that we must hesitate to consider the arrested case of tuberculosis as eligible for standard insurance—not until after many years of uninterrupted good health anyway.

We have a lot more to learn before we can accurately measure these after histories. So much comes into consideration, the ages at which the patients go to the sanatoria, the nationality or stock which they come from, the kind of occupation after they leave the sanatoria, whether a favorable occupation or not, etc.

Heredity

Dr. Bradshaw—

In an article entitled "The Hereditary Factor in the Etiology of Tuberculosis" Dr. Alber Govaerts summarizes a biometrical and statistical study of 214 families with 185 tuberculous matings and 29 non-tuberculous matings. He offers the following conclusions:

1. The percentage of tuberculous offspring is higher in tuberculous families.
2. In the same surroundings there is a greater occurrence of tuberculosis without than with close contact.
3. There is a definite parental defect introduced.
4. The maternal influence is very slight and there is no evidence of prenatal influence.
5. From a eugenic standpoint it is not advisable for two tuberculous subjects to marry.

Dr. Beckett says—"I believe it is a well established fact that any individual who has a tuberculous family history as a rule is more susceptible to the development of that disease at any age than is an individual who has no such family history."

Dr. Krauss in a recent article entitled "A Few Observations on Immunity" amplifies Dr. Beckett's statement in a very interesting manner. He cites the following experiments.

Animals that were entirely free from tuberculosis and animals that had been rendered immune either by vaccination with increasing doses of tubercle bacilli or tuberculin or others that were immune because they had actually recovered from some clinical manifestation of tuberculosis were used. These animals were inoculated with virulent acid-fast bacilli and in each instance a body response of such uniformity took place that the observer could well speculate on the processes of immunity and infection or reinfection.

In the non-tuberculous animals it was evident that the reaction was slow and that the infectious process evolved indolently in the body, the animal remaining meanwhile in normal health. It was uncommon to discover visible evidences of infection during the first ten days even after the application of large numbers of tubercle bacilli. Noticeable illness began only two or three or more weeks after the disease was well established. These were healthy, non-immune animals. Eventually they succumbed to their infection which became extensive and widely disseminated.

Immune animals, when infected behaved in a totally different manner. In their case infection really meant reinfection. The inoculation of virulent bacilli always precipitated an immediate tissue response. Within a few hours the points of bacillary focalization became inflamed, the severity and type of response varying with the numbers of bacilli concerned. At the same time the animal became rapidly and acutely ill. Yet these immune animals, except those receiving massive doses of bacilli, did not die. Tubercles developed rapidly but later healed.

How do these experiments contribute to a better understanding of the disease in man? In the animal of experiment it was always the immune animal that became quickly and profoundly ill with acute tuberculosis. Never the animal infected for the first time, no matter how large or virulent the dose.

The conclusion which must be drawn is that the immune animal is acutely ill because of its immunity. Applying this reasoning to the man acutely ill with tuberculosis we realize

that the man struck down with pleurisy with effusion, or tuberculous pneumonia or generalized miliary disease is "going bad" not because, as clinicians have learned to say, his resistance is low, but because he is an immune animal that has suffered re-infection. For us, the lesson from this in Life Insurance Medicine is that the man supposed to be immune from heredity or previously cured infections is always ready and subject to this violent, sudden reaction from re-infection and in man it is the virulence of the reaction rather than the severity of the reinfection that may be fatal. Dr. Krauss simply states these facts; he does not offer any conclusions.

Increased Mortality of Tuberculous Individuals

I agree with the statement showing the increased mortality of those who have had tuberculosis. Dr. Beckett quotes statistics from the Sanitarium of the Modern Woodmen of America and the Trudeau Sanitarium at Saranac Lake. At Trudeau of the 814 patients traced, over a period of 20 years, 666 had died and of these 160 or about 19% had died of some form of tuberculosis.

Some years ago, in a personal communication to Dr. Williams, the present director of the National Tuberculosis Association, Dr. King late director at the Loomis Sanitarium, said that his experience was that all of his patients ultimately died of tuberculosis with the exception of one man and he was run over by a train on his way back to the Sanitarium because of a relapse.

The Mutual Life statistics are from a small group because we accept few cases that have actually had tuberculosis.

In those in which there was a history of blood spitting without finding pulmonary involvement on examination, 11% died of tuberculosis.

A history of tuberculous glands or scrofula $33\frac{1}{3}\%$ died of tuberculosis. History of tuberculosis of bones or joints, $33\frac{1}{3}\%$ died of tuberculosis. History of pulmonary tuberculosis over 10 years ago, 50% died of tuberculosis.

This gives us in all cases in which some evidence of previous

infection was manifested, an average in the group of 31% dead of tuberculosis.

Dr. Rogers in a summary of three similar groups studied shows an average of 36% dead of tuberculosis. Both studies showed a higher mortality in the first five year groups and in the younger ages.

Effect of Gas

Sufficient time has certainly elapsed to estimate the effect of being gassed in the war. Those who were gassed, but have fully recovered I believe are safely insurable. A recent paper by Hankin and Klotz, summarizing the soldiers who were gassed and now quartered at the National Soldiers' Home in Tennessee throws light on this subject.

Their experience is that a gassed soldier remains a sick man; that his symptoms and physical signs continue and he shows little or no tendency to get well. The ones that recovered, did so within the first few weeks and have remained in good health. The most constant pathological condition found was chronic bronchitis. Tuberculosis also was slightly more prevalent.

Dr. Beckett's summary of the type of case that is acceptable for insurance is admirable. We must honestly admit that they are all substandard, but some are insurable within strictly drawn lines. I do not believe that anyone who has ever had a positive sputum or who had demonstrable physical signs, even though they are of a healed lesion, at the time of examination is insurable at standard rates. The importance of bone and gland tuberculosis and the other tuberculous manifestations decreases with time. A man of standard physique living a normal life 10 years after a mild or questionable infection is not infrequently insurable for a small amount at standard rates.

In view of the general knowledge and good instruction the laity now has about tuberculosis, it is possible to reduce the age beyond which cases in which exposure is the impairment from age 40 to age 35, and in some instances to age 30.

I heartily second the idea of a National Department of Health of which the Cabinet Secretary should be a physician.

Dr. McMahon—Dr. Kennon Dunham, of Cincinnati, has very kindly prepared a paper which he will read at this time in connection with the discussion of Dr. Beckett's paper. Dr. Dunham's subject is the value of X-Ray Chest Examinations in Life Insurance work.

A PRACTICAL AND VALUABLE METHOD OF X-RAY
CHEST EXAMINATIONS FOR THE APPLICATION
IN LIFE INSURANCE.

BY KENNON DUNHAM, M.D.

Director, Cincinnati Tuberculosis Sanatorium, Professor Tuberculosis, University of Cincinnati. Cincinnati, Ohio.

The object of this paper is to interest your organization in the use of X-ray plates for the purpose of your obtaining more exact knowledge of the thorax of your applicants. I hope to show that the valuable knowledge thus derived more than justifies the effort and expense; that a simple method of securing and interpreting these plates has been worked out; that almost unbelievable information can be obtained and that there are grave dangers to be avoided, but that ordinary intelligence can do this. I can state that the Union Central Life Insurance Company has tried this method and finds it valuable.

Life insurance is a business; medicine is an art; both are based upon the sciences, both were primarily conceived to serve, yet both must be placed upon a firm financial basis or they cannot endure. Let us therefore consider cost at once. Then while I am talking you can determine whether the value is worth the price. A few stamps and fifteen dollars, expended for the taking, reading and transportation of stereoscopic chest films will bring this information across the desk of anyone of you who cares and who will take a little trouble.

Two processes are required: the taking of plates and the interpretation of these plates. Let us call the man who is to

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take the plates the roentgenologist and the man who is to interpret these plates the reader.

There is an experienced roentgenologist in almost every town where you have an important agency. His services can be easily secured. The reader who is to interpret these plates for you in a uniform manner and upon whose judgment you are to base your risk is another matter. He must know how to take and interpret a complete case history, be an anatomist and know the gross and microscopical anatomy of the lungs, be a pathologist and understand the pathology not only of pulmonary tuberculosis but all other diseased conditions of the chest; know thoroughly the physical examination of the normal chest in order that he may be able to interpret the examination of a pathological chest; have a thorough understanding of the pathogenesis of tuberculosis; must know the relationship of resistance to prognosis. Then as an X-ray man he must apply his knowledge of anatomy to the X-ray of a normal chest and his knowledge of pathology to the X-ray of a pathological chest, and thus be able to make a complete differential diagnosis of all chest conditions.

The completed problem is now presented to you after more than fifteen years of constant effort which has included special study of electrical and mechanical engineering, research in anatomy, pathology and roentgenology, a careful comparison of physical signs and X-ray densities, constant study of clinical medicine and the many phases of tuberculosis. The results of this research have been carefully checked by gross and microscopic pathological study of hundreds of autopsies, the lungs of which have been X-rayed both before and after death. By this method X-ray densities have been translated to cellular pathology and the results of this work have been tried in practical insurance examination. Only time and a statistical study of thousands of cases can fully prove or disprove its value to you.

I desire to show what this method will accomplish and I ask you to estimate its value. Think of it as the missing link for which you have been searching to complete the chain of evi-

dence. The links of personal and family history and habits and mode of life are now joined by the X-ray to physical examination and personal appearance. You can understand the force of this truth where you know that much of the past history of every man is indelibly written upon his chest plates. One must study the bones, the diaphragm, the heart, the aorta and pleura and the nodes.

The reader should be given the age, the race of the applicant and the X-ray technique by which the plates were taken. Given these facts your reader can detect for you the effects of an old pneumonia, an empyema, pleurisy, syphilis of the bone or healed tuberculous nodes. These latter even produce resistance. We can diagnose tuberculosis, unresolved pneumonia, abnormal lung exudates such as syphilis, Hodgkins' disease, sarcoma, carcinoma, aneurism, dilated or elongated aorta, arteriosclerosis, enlarged heart and substernal goitre.

Given a questionable history of tuberculosis and a clean X-ray plate the applicant should be accepted without further question of pulmonary tuberculosis, because tuberculous scars are left as indelible marks which will be changed by time but never erased. Many times rales in the chest suggesting tuberculosis are due to infected sinuses. If the examiner has found definite rales and the plates show no evidence of pulmonary tuberculosis, the sinuses, especially the maxillary, should be suspected and the case held for further observation. Such applicants should not be summarily rejected.

The examiner often would not suspect sinus trouble even were he making the examination for the patient instead of the company. The patient may have no symptoms nor complaints except a susceptibility to colds. Such a case with negative X-ray plates is almost invariably the result of sinus infection.

Tuberculosis which until recently claimed one-tenth of our deaths and earned the name of the "Great White Plague" is still with us. The unexpected drop in the death rate during the last two years is very gratifying but the fight is far from being won. Tuberculosis has many aspects and I wish to stop and consider a few of them because the X-ray chest plate will

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tell you more about what was, is and will be, with regard to tuberculosis in a given chest, than all other methods combined.

We know that the military age 20 to 30 is also the tuberculosis age. The cases develop quickly during this period; often without previous history or symptoms. We know that after 40, the chance for a winning fight and victory is good. I wonder if it is as well known that after 50 and especially after 60, healed tuberculous lesions are frequently reactivated and the chances of recovery are poor.

You have a large mass of statistical proof which shows that a case of tuberculosis healed for ten years is a good risk. The weakness of this argument lies in the fact that you have no proof that all of your diagnoses were correct. I am sure that they were not. The reason for this conclusion is that at the end of the year you have amassed a large number of mistaken diagnoses. If you had limited your study to cases having had tubercle-bacilli in the sputum or cases having definite tuberculosis in the lung, you would not have jumped so quickly to the conclusion that a tuberculous lesion is safe after ten years. Possibly fifty per cent. of them break down.

The X-ray will be of great value in weeding out these cases. It will show which are safe risks and which are not. But if you make the mistake of concluding that cases showing definite old tuberculous lesions in the lung proper are good risks because they have a history dating back ten years or more you will soon see the errors of your statistical study. They should not be accepted as first-class risks unless the lesion is very limited and well walled off.

About three cases of tuberculosis developed in every thousand healthy men admitted to the old army before the Great War. It is probable that that per cent. cannot be much lowered by any form of examination, and that this number developed the disease after the examination. Your rates easily cover this loss. What is more important to you is the number of men who are being excluded unjustly because you are afraid of these 3 per thousand. A careful X-ray examination will allow you to accept as good risks many men who are today

being excluded. This will be good for the country, the man and the agent and the company. I wonder if you can estimate the number of men and women who are being denied insurance unjustly because of the fear of tuberculosis. It would be most valuable knowledge. Allow the X-ray to eradicate this injustice and the relation of the agent and the medical director will be greatly improved.

The problem can never be solved by a physical examination. Gentlemen, the limitations of a physical examination have never been written. You men probably know this better than any other set of men. I wonder if you are not blaming your examiners for what is not their fault but the fault of our system of teaching which gives physical signs more value than they deserve. A systematic use of the X-ray should make better examiners. Many life insurance examiners are given a high rating by you because they are prompt and make a good report in a legible hand. Check a few of their histories and physical examinations with a good X-ray report and you will be able to estimate the value of these examiners. This is especially important when the examiners of different companies have rendered conflicting reports.

At the Cincinnati Tuberculosis Sanatorium where now we systematically X-ray every case upon admission the results have been nothing short of amazing. We now know that we can have cavities without physical signs to even suggest it; that we can have tuberculosis without rales; that broncho vesicular breathing may mean much or little; that we often suspect fluid without it being present; that fluid can exist without it being detected. I could interest you in this subject for an hour. The important point is that an accurate history, intelligently interpreted, a good physical examination given its proper weight and an intelligent X-ray report will give you Medical Directors a foundation upon which you can base your risks.

The accuracy with which adult pulmonary tuberculosis may be detected is beyond belief. An X-ray report should accurately locate such a lesion, describe its character and extent and

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classify the kind of tuberculosis. Frequently its activity can be determined and a prognosis accurately made giving the approximate number of months a patient may be expected to live. As I have said such reports have been routine at the Cincinnati Tuberculosis Sanatorium for years. They are criticized by an able House Staff and an average of about 70 autopsies each year. If these reports were based only upon the ego of the reader, they would have been laughed out of court long since and the expense discontinued.

Let us now consider how this can be accomplished and what dangers must be avoided. I have said that you would require a roentgenologist near each important agency and one reader. A contract could be made with the roentgenologist to take the plates and forward them to your reader. The technique of taking and mailing the films should be standardized by the reader. The reader will ascertain the character of the equipment available and know the possibility of each laboratory. He can dictate his technique accordingly. The roentgenologist can make a small charge for he is required to give no opinion and the work might be done by a technician. Thus you will secure standard technique and uniform readings and soon have sufficient data upon which to base an accurate judgment of the method without any outlay of money.

The only danger from which you need protection is your reader. He determines the technique and interprets the findings and if these readings are not based upon sound knowledge and properly taken plates the foundation of your house gives way and the structure falls. Given a reader who will demand proper technique and who has the required knowledge and every company may safely expand its business by using this method.

I have not taken your time to describe how these conclusions may be reached but I do wish to call your attention to a few mistakes which are commonly made and which would cause you to accept or reject the wrong applicant.

Let us consider some errors of technique and readings, which might cause you to accept bad risks.

If plates are over exposed you lose much detail and active tuberculous exudates may be overlooked. If your reader does not understand that tuberculous exudate in early cases shows only delicate fans he will overlook the most serious lesions. In either case you will accept a bad risk. If he believes that single plates are as good as stereoscopic plates he is only seeing the gross lesions and the director must consider all rales as tuberculous or he will accept bad risks. By stereoscopic plates we quickly determine which rales are due to tuberculosis.

If the patient breathes during exposure anything may happen and the plate should not be read unless the lesion is massive. In such a case you have trouble and expense without results. If the amount of air in the lungs at time of exposure is not average your reader will suspect emphysema or a high diaphragm. Such would be misleading but not serious.

Let us now consider errors of technique and reading which would cause you to reject good risks. You will see that they are the more numerous. If the plate is overtimed or the patient breathes between exposure, it frequently happens that the appearance of a fan is artificially produced suggesting recent lung exudate and your reader must conclude that you are dealing with active inflammation of the lung possibly tuberculosis. If the angle of the principle ray is not correct the thoracic viscera will be distorted and the reader may diagnose an enlarged heart or aorta or conclude that a slight pleurisy has serious significance. If the reader does not know his anatomy he will not know where the pulmonary nodes are located and may mistake a calcified lymph node for a lung lesion which should be expected to break down. Such lesions within a node argue for immunity when in the parenchyma of the lung they often break down. If he does not understand that many lung infections such as, broncho pneumonia or measles, produce heavy trunks, he will call the case peribronchial tuberculosis and you will lose a good risk. If he believes that a slightly thickened pleura at an apex is of necessity due to tuberculosis he will have you reject a man many years after he has recovered from pneumonia.

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These are a few of the errors of technique and reading which could cause you to discredit this most valuable laboratory aid.

I have hesitated to emphasize the dangers which faulty technique and poor reading make possible. I feared that you might not understand that these dangers have been surmounted. You as intelligent men must know that any departure from the beaten path must lead to rough going. I believe that by facing these dangers frankly I may the more easily challenge your interest.

I now hasten to say that we have overcome these dangers by securing accurate technique and intelligent readings. This is why so much time has been spent in telling you of the study necessary to master the subject and why so much emphasis has been placed upon the selection of your reader. If he is competent and conscientious you need have no further worry. He will criticize the technique and render your readings upon which you may safely base your risks.

In these few remarks I believe that I have shown you a simple economical procedure which will enable you to increase your business by eliminating poor risks which you are now accepting and including good risks which you are now rejecting.

Dr. McMahon—Dr. Pauli will discuss this very valuable contribution.

Dr. WILLIAM O. PAULI: When we suggested to our President that we would like to have presented a paper on the value of X-ray examination in life insurance, we did so with the idea of harmonizing the requirements of life insurance companies in general. Agents seriously object to any requirement, when other companies with an equally favorable mortality rate do not request such a rigid examination.

It is the purpose of this Association to have Medical Directors get together for our mutual benefit in the selection of risks. If we can show that the X-ray examination, in certain selected cases, is justifiable and have more companies take an interest in X-ray examination, then we will have planted the seed for further progress in medical selection. It usually takes

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ten years from the time that a new method is introduced in medical science before it is generally adopted.

The agents might say that in the early days we did not have blood pressure or urinalysis at the Home Office or the sugar test meal or a Wassermann test, yet the mortality was favorable. Are you trying to drive the agents out of business by requesting an X-ray examination?

We had a difficult time educating agents to the advantages of Home Office urinalysis and it took many years before other companies started Home Office laboratories. Our esteemed colleague, Dr. Fisher, had the same trouble with the blood pressure test, and the agents have survived. They do balk on the sugar test meal and we are having our troubles on this score at the present time.

But if we advise the agent that a risk can not be considered unless an X-ray examination is submitted, he often tries to place the insurance in another company, that does not require X-ray examinations. We all know that agents will broker an application and select against the Company, if they find the gates wide open.

We do not intend to have X-ray examinations on all cases, but only on certain cases. These selected cases are as follows:

- (1) History of pleurisy with effusion during the past five years.
- (2) History of blood spitting during the past ten years.
- (3) History of impaired lungs on previous examination, with M. I. B. record of 407x.
- (4) History of a change of residence on account of health, with a doubtful diagnosis.
- (5) Lightweights with a history of definite exposure to tuberculosis.
- (6) Lightweights who are under thirty years of age, who apply for a large amount of insurance.
- (7) All cases where the examiner finds the lungs impaired before the risk is finally rejected.
- (8) It can be used to establish a diagnosis on early cases of tuberculosis, who submit a claim for disability benefits.
- (9) It can be of service in our Health Test conservation work, especially for large policyholders.

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An X-ray examination will detect a previous history of tuberculosis, which the applicant has concealed in order to obtain the insurance. Applicants can easily conceal a history of blood spitting or pleurisy with effusion, but they can not conceal the scars revealed by an X-ray examination. A history of tuberculosis does not have the same significance today in the selection of risks that it had twenty years ago. We all know that the diagnosis of tuberculosis is made frequently in the early stages and treatment restores them to good health. There are many more cases of latent tuberculosis who apply for life insurance at the present time. There is also an important group of arrested cases, who give a previous history of latent tuberculosis.

We can differentiate and select these cases only by an X-ray examination, interpreted by a specialist who is an expert in this line of work. If we do not have an expert interpretation of the X-ray film, we may be misled by an erroneous diagnosis, which is worse than no X-ray examination. We always discard a poor X-ray film, which is the result of bad technique.

When we request an X-ray examination, we authorize our agent to have an X-ray examination of the chest made by a specialist recommended by our chief medical examiner in the home town, for which the Company will pay a fee of \$10.00. The X-ray picture is then submitted to the Home Office and referred to Dr. Dunham for his interpretation. The Company pays Dr. Dunham a uniform fee for each report. We also receive a report from the roentgenologist who made the examination, but this is not essential. It has been our experience that these reports enable the Company to issue insurance at standard rates to many risks which would otherwise be declined. On cases declined by other companies because of tuberculosis of the lungs suspected, we require an X-ray examination of the chest and if the X-ray examination is negative, we approve the risk at standard rates.

This is the first opportunity we have had to discuss this subject. There is a very interesting paper by Dr. Lawrason Brown and Dr. L. G. Cole which was read by title at our meet-

ing held in 1920 and printed in the proceedings of the Association. If any of you have not seen this article, I would suggest that you do not fail to read it. This subject is too important to be buried without some expression of opinion from our members. I know that other companies are also using X-ray examinations in certain cases, and I would like to have them voice their opinion of the feasibility and value of X-ray examinations in Life Insurance.

We are very fortunate to have Dr. Dunham present his views on the value of the X-ray examination of the chest, as pertaining to life insurance examinations. Dr. Dunham has made a special study of the subject for the past fifteen years and has published at various times the results of his researches on the interpretations of the various densities revealed in the X-ray plate of the lungs. Dr. Dunham is well qualified to speak upon this subject, as he receives X-ray plates from roentgenologists from almost every State in the Union, for his interpretation of the findings. In the majority of these cases, the roentgenologists themselves were doubtful about the diagnosis and desired the opinion of a man who has had probably more experience in this line of work than any man in this country. So I wish to extend to Dr. Dunham my sincere thanks for coming before this meeting and presenting such an interesting paper before this Association.

Dr. Beckett—I tried in my paper to be very conservative in the statements I made. I am very glad to hear Dr. Knight say that the drop in tuberculosis is much greater than I have stated. In fact, the majority of authors that I consulted made much broader statements than I quoted.

I think it is important that cases should be treated in a sanitarium as these cases are better risks than those who are treated outside of a sanitarium, and I also believe that most of these cases are not treated for a long enough time. An agent will tell you that this man had a slight attack and was treated for only two or three weeks or a month; I should prefer to hear that he had been treated for six months or a year.

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With regard to re-infection, I was glad to hear what Dr. Bradshaw said with reference to this point. I remember talking with Colonel Bushnell of the Fort Bayard Sanitarium, some years ago. I asked him if any of his cases whom he had dismissed as cured ever came back. "Oh yes," he said, "Many of them. They get out, neglect themselves, have relapses and come back." I said, "What percentage of them are cured after the relapse?" "Oh," he said, "They all die."

I believe education is probably doing more than anything else to reduce the disease of tuberculosis. It has been only recently, you might say, that the State and Government have been giving us the help that they are extending at the present time. I remember not a great many years ago, we made an effort in California to get the Legislature to give us a sum of money for publishing literature to send out to the people, and especially those afflicted with tuberculosis. After considerable effort we got the munificent fund of \$1,500 allotted to us for that purpose, and then our Comptroller said it was illegal and he wouldn't let us have it! But to-day our State is dealing with us generously and the effects are seen in the report of the health officials of the State.

Dr. McMahon—Dr. H. B. Anderson will now present to us his paper on Focal Infections. I happen to know that Dr. Anderson has given a great deal of time and study to the preparation of this paper and I am sure we will find it of very great interest.

CHRONIC FOCAL INFECTION OF THE MOUTH AND
TONSILS IN RELATION TO LIFE INSURANCE

BY H. B. ANDERSON, M. D.,

Medical Referee, Imperial Life Assurance Co.

Investigations during recent years have established the importance of chronic focal infections as causes of many serious systemic diseases, at times shortening the expectancy of life or leading to disability. These infections are usually of a low grade of virulency, often latent or producing little local discomfort; yet under conditions of lessened systemic resistance induced by exposure; nervous or physical overwork; under-nutrition or dietetic indiscretion, alcoholism and other excesses; acute diseases as pneumonia, or influenza; constitutional diseases as gout and diabetes; following parturition or operations; and in advancing years, there is a liability for the infection to invade the circulation and thus produce constitutional disease. Chronic focal infections may also be a source of mixed infections occurring with other diseases, as pulmonary tuberculosis, in the streptococcus pleurisies or pneumonias of influenza; the secondary invasion at times over-shadowing the primary infection in point of seriousness. Focal infections do not give rise to an active disease with a definite symptomatology and course proceeding to recovery or death in accordance with the efficiency of the various factors included under the term systemic resistance, but the cause often remaining sealed in, the effects may be shown by a variety of clinical manifestations, appearing and subsiding over the period of years during which the local foci persist.

In some cases at least they are responsible for the persistence or recrudescence of acute manifestations or the progressiveness of certain cases of chronic disease such as nephritis, endocarditis, myocarditis, gastric and duodenal ulcer, gall-bladder infections, etc. They certainly tend to aggravate such conditions as diabetes and Graves disease and may be a cause of

general nervous disturbance or more serious organic nervous and mental disorders.

One cannot read Beard's original monograph on neurasthenia without being convinced that many of the symptoms which he described as characteristic of that disease were really such as would now be attributed to focal infections. He evidently confounded effect with cause in enumerating decay of the teeth as one of the *results* rather than as a cause of neurasthenia.

The introduction of arsenic by Spooner of Montreal in 1838 as a devitalizing agent for the dental pulp, unintentionally initiated in certain common dental procedures perhaps the most far-reaching experiments that has ever been carried out on human beings. By these means a new set of conditions was set up and, as one would expect a new series of disease-reactions resulted. In the light of knowledge acquired during the past few years, aided by the application of the X-ray to dental diagnosis and by bacteriological and other clinical methods, this experiment may now be summarized:

Teeth, the peridental membranes or pulps of which had become infected by *streptococcus viridans* and which, by reason of the resulting pain, would theretofore have required extraction for relief, had their pulps destroyed by arsenic and the necrotic material cleared out. In this way, the nerve—Nature's danger signal, was removed, as well as the chief source of the tooth's nutrition, viz., the vessels of the pulp. The resulting cavity was filled with some inert substance or the teeth were crowned, buttressed by bridges, or perhaps pivot-teeth inserted into devitalized roots. This resulted in a large percentage of teeth thus treated, in sealing up in the pulp cavity, dental canals or at the apices of the teeth, of a streptococcus infection of a low grade of virulency, which usually remained there for an indefinitely long period. The unsuspecting individual was thus exposed to the effects of multiple points of streptococcus infection around what were virtually infected foreign bodies (dead teeth) embedded in the living tissues of the alveoli. Is it therefore a matter for surprise that a series

of local and systemic reactions should develop in response to so potent a cause of trouble or that symptoms should arise, not all at one time, but at intervals during the long period of exposure?

I have complete records now of nearly 2000 cases which I have carefully reviewed in the hope of deriving certain data which might have a bearing upon the question of focal infections in relation to life and disability insurance. While the study of these cases of oral and tonsillar infection and their interrelationships with other clinical conditions has served to impress one with their importance as causes of serious diseases, many difficulties arise when one attempts to arrange the data in a statistical form desirable for insurance purposes. The completely edentulous for instance would no longer be classed as cases of oral infection and yet such individuals evidently had suffered from serious oral infection and its attendant dangers. Again the universality of some degree of oral infection in our population would necessitate one's emphasizing more the *degree* and *character* of the infection rather than its mere presence. For this reason therefore the cases were classified as to severity as x, xx, xxx, xxxx, and the tonsillar cases were similarly designated

As focal infections of the teeth and tonsils are both recognized as causes of transient albuminuria and also of more serious renal disease, albuminuria was selected as a type of impairment upon which the study might throw some light. In this connection an interesting point appeared in that albuminuria was found in 32.4% of all the cases of oral and tonsillar infection occurring together, examined in 1919; 21.4% in 1920; 12% in 1921; and 9.2% in 1922. Precisely the same methods of examination were followed in these years, so that the decreasing incidence from 1919 onward is probably related to the decreasing frequency of serious epidemic diseases during that time.

As the cases generally were of the chronic ambulatory class seen in office consultation, only a small percentage of which had suffered from the prevalent epidemic diseases, some other

factor would be required to explain the marked yearly variations. No doubt the variation is partly explained by the lessened general morbidity and mortality rates during the past three years, but it is possible in times of epidemics such as influenza, that these foci of local infection are more frequently stirred into activity.

In regard to the relationship of albuminuria to the *degree* of oral infection in 1919, 1921 and 1922, x showed it in 27%, xx in 26%, xxx in 28.2%, and xxxx 32%.

The tonsillar cases during the same three years showed very similar figures but of course the two conditions frequently co-existed: x, 27%; xx, 26% xxx, 30%; xxxx, 39%.

Thus the severer grades of infection in both show a considerably increased incidence of albuminuria but the difference is not sufficiently great or the number of cases (1393) large enough to warrant one in drawing conclusions that are more than suggestive.

In 1919 alone the percentages are: x, 39%; xx, 38%; xxx, 54%; xxxx, 53%. It is thus shown that not only was the percentage of cases of albuminuria greater in 1919 but that the severer grades of oral infection showed a relatively higher incidence of albuminuria than the lower grades than in the 1921-1922. This might suggest that when the foci of infection are stirred into activity by some general cause as in epidemics of influenza that the severer grades are relatively more potent in producing albuminuria than the milder grades. These figures obviously have not the same significance that they would if based on the class of the population applying for insurance, yet I present them for what they are worth in the absence of more definite data bearing on the question.

In nearly a thousand serial cases, plate cultures on blood-agar were made from the tonsils in order to determine the bacteria associated with chronic tonsillar infections but eventually this laborious procedure was given up on being convinced that its clinical value did not warrant the expenditure of time. Radiographic examinations of the teeth were made as a routine and in many cases cultures were made from the apices

at the time of extraction and the appearance of the extracted teeth noted. The *s. viridans* was the organism almost invariably recovered from apical infections.

Not infrequently tonsillar infection has been found in the absence of or with only slight oral infection, but in cases of severe oral infection the tonsils were practically always diseased.

In regard to radiographic examinations, while they have undoubtedly been of great value in the study of oral infections they do not furnish the most conclusive evidence of the presence or seriousness of infection and if too much reliance is placed upon them they are often misleading. It is well known that the most serious forms of infection may exist with little or no radiographic evidence of its presence. My own experience has led me to the conclusion that a consideration of the pathology of the condition, the history of the case and a careful observation of the teeth and gums, along with the clinical data pointing to involvement, afford by far the most easily accessible and reliable evidence of active infection. I am also convinced that the physician is better qualified than the dentist to determine the presence of infection of importance from the viewpoint of systemic disease and therefore it is unfair for us to place this responsibility on the dentist.

Similarly in regard to the tonsils—a history of recurrent attacks of sore throat, tonsillitis or quinsy, the coexistence of extensive oral infection, the presence of redness and hypersecretion of the fauces, tonsils or pharynx; of atrophic adherent or cryptic tonsils from which pus or caseo-purulent matter can be expressed by an instrument or the fingers; the presence of stumps or glazed-over remains of incomplete operations and tender or enlarged sentinel lymph-nodes about the angle of the jaw, with a history of past, or the existence of present clinical manifestations of systemic infection, enable the experienced examiner in the large majority of cases, after a few minutes investigation to determine whether a clinically active and therefore potentially dangerous infection exists. This is important from the insurance standpoint, as more

specialized and expensive investigations would not be feasible in the ordinary routine examinations of applicants, whereas the clinical evidences of infection above noted are readily to be ascertained by any ordinarily competent examiner. My own experience accords with that of others in the conclusion that recurrent attacks of sore throat, tonsillitis or quinsy with consequent adhesions, occlusion of the crypts, fibrosis and atrophy of the tonsils, furnish practically conclusive evidence that a persistent, clinically-active and potentially dangerous infection of the tonsils exists, which cannot be cured by measures short of complete enucleation.

If there is a past history or present evidence of any of the systemic diseases ordinarily recognized as due to or aggravated by focal infections, these more significant manifestations should also be provided for in making the selection. It should also be borne in mind that when a patient has become sensitized to an infection as that of the *s. viridans*, which does not stimulate a systemic reaction with the formation of antibodies, that a degree of residual infection which originally might have been insufficient to have produced trouble, may be potent in causing a recrudescence or maintaining existing systemic disease.

While applicants for insurance with such a history or such a condition found on examination, may not show any impairment in the general health for long periods when the patient's resistance is good, yet I do not think they are entitled to insurance at the standard rates.

In considering oral infection from the pathological side it is well to remember that ordinarily it is infection of the pulp which causes the death of a tooth, and that when the dentist removes such pulps and treats the canals, even by the most approved technique, he is dealing with tissues already infected, the viability of which further is compromised by the removal of the pulp, including the nerve as well as the vessels furnishing the chief source of the tooth's nutrition. While a certain amount of nutrition is supplied by the peridental membrane this structure is often involved by extension from the apex

or coincident pyorrhœa, so that such a tooth is virtually a dead, infected, foreign body.

The investigations of Price, Rhein, Davis, Duke, Black, Talbot and others, have shown that 80% or more of devitalized teeth are, or soon become, the seat of periapical infection. Talbot goes so far as to say that treating and filling roots results in failure in 95% of cases and in the other 5% the periodontal membrane becomes diseased and the tooth eventually a source of infection. This necessarily applies to large fillings where the pulp has been destroyed, to pivot teeth and to devitalized crowned teeth. It of course does not include vital teeth, crowned at times to serve as the abutments of bridges or to support dentures. Carious teeth with exposed pulps and old roots, are always the seat of infection, though not being sealed in, as with the former, there may be a better chance for drainage and therefore less danger.

If we accept the conclusions of the authorities before mentioned therefore, devitalized crown and pivot teeth are really foci of infection by the *s. viridans* in from 80 to 100% of cases. This being the case it is only necessary for the examiner to count such pulpless or devitalized teeth in the applicant's mouth in order to inform the medical referee with approximate certainty of the number of foci of infection to which he is exposed. If in addition the examiner furnishes information in regard to the local appearances presented and the evidences, if any, of systemic involvement, these along with the data contained in the routine examination of the applicant, should enable the head-office medical staff to form a judgment as to the extent and activity of the oral infection and its bearing on the selection of the risk. If one considers devitalized crown and pivot teeth as representing foci of streptococcus infection and especially if there are many of them, and if the local and general examination further shows evidence of activity, suggesting that the limits of the individual's resistance are being approached, it does not appear to me that the applicant is entitled to insurance at standard rates.

The appalling frequency of dental infection is shown by

the investigation of the teeth of school children in various centers:

New York City	885,577	cases examined	caries found in	524,356	or nearly	60%
Detroit	92,000	"	"	defective teeth found in	98.90%	
Milwaukee	26,285	"	"	" " " "	23,173	
Chicago	33,381	"	"	caries " "	30,044	
Toronto	49,081	"	"	" " "	95%	

We are also aware of the large proportion of the adult population who have devitalized teeth. Tonsillar infection is also extremely common being present in greater or lesser degree in nearly 60% of my own chronic ambulatory cases.

It may therefore be the view of many that when so large a percentage of the insurance population is thus affected that the impairment is provided for by the general rates of insurance and that a special rating is neither necessary nor feasible. One is prepared to concede that this applies to the larger proportion of cases which fortunately consist of the minor and less dangerous degrees of infection. As we approach the outer limits of this group, however, there is a smaller one, wherein the number of foci is greater, representing the more dangerous periapical infections of devitalized teeth, or recurrent tonsillar infections, where the applicant's appearance and physique are less satisfactory, where there is a history of past minor ailments or definite diseases such as digestive disturbance, nervous disorders, rheumatism, endocarditis, perhaps transient or recurring slight albuminuria, etc., in which our present knowledge of the pathology and the clinical aspects of focal infections, is sufficient to warrant our constituting them a special class in which provision should be made for a higher mortality by a rating. Such a rating should be conditioned not only on the nature and degree of the previous illness or ailment but on whether an efficient cause for its persistence or recurrence, such as focal infections constitute, still exist.

In other words a number of impairments in themselves perhaps of minor importance should be considered from the

viewpoint of the existence of a persistent, efficient cause, of which such impairments are not only symptomatic but also the early danger signals of more serious conditions prone to develop later on.

For this reason I believe it would be well to issue instructions to examiners and to formulate questions to elicit more definite information in regard to recurrent tonsillar infection and the evidences of its persistence and activity and in regard to oral infection, of the number of devitalized, crown, pivot or stopped teeth and other evidences of local and systemic infection before outlined.

The question naturally arises, what one's attitude should be where the focal infections in teeth and tonsils, supposed to be the cause underlying the impairment, have been removed. While such procedure may eliminate the original cause, a sufficient lapse of time only can determine whether the infection which has already gained a foothold will subside and whether secondary foci in the lymph glands or distant organs or tissues will clear up or if permanent organic mischief has already been produced.

Is it not considered feasible at present to make provision for a class of oral and tonsillar infections calling for a special rating? The precise statistical information obtained in this manner before suggested would serve as the material basis for future action.

By means of such information we should in time be better enabled to evaluate certain impairments, such as transient, slight, recurrent albuminuria of adults, in themselves unimportant but significant as possible early danger signals of organic renal disease. The advantage of substituting as far as possible a definite etiological, in place of a doubtful symptomatic basis for the selection of risks would enable us to discriminate with greater accuracy. I wish now to report a common type of case wherein a precise knowledge of the presence or absence of oral or tonsillar infection would be of material value in making a selection.

Reported as follows:

- Oct. 18, 1912—Albuminuria found on examination.
Presence of casts found on examination.
- Dec. 24, 1914—Albuminuria found on 2 separate examinations.
Presence of casts *not found to exist* (Oct. 18, 1912).
- Dec. 9, 1918—Functional cardiac trouble found on 2 separate examinations.
Presence of casts *not found* on examination.
- June 6, 1921—Persistent rapid pulse between 100–110 and over 100, found on numerous examinations.
A small amount of albuminuria found on examination, intermittently.
Presence of casts *found not to exist* on numerous examinations Dec. 9, 1918.
- April 30, 1922—A moderate amount of glycosuria found on examination, intermittently.
Hematuria found on examination, intermittently.
Mitral regurgitation found on examination.
Hypertrophy of heart found on examination, a moderate degree.
Persistent rapid pulse between 90–100, and over 110 found on 2 separate examinations intermittently.
Albuminuria *found not to exist* on 2 separate examinations (June 6, 1921).
- July 31, 1922—Hypertrophy of heart *found not to exist*.
Hematuria *found not to exist*.
Mitral regurgitation *found not to exist*.
Glycosuria *found not to exist* on numerous examinations.
Urinalysis made 15th June, 1922, no albumen, no sugar, microscopic negative; specific gravity 1.027.
Test meal, 28th July, 1922 (ordinary meal heavy in carbohydrates) specimen taken two hours afterwards, no albumen, no sugar; microscopic negative.
Specimen examined 29th July, 1922, no sugar; no albumen; microscopically negative.

This represents a type of case where the history of cardio-vascular-renal symptoms recurring at intervals over a

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period of ten years is suggestive of a persistent cause such as chronic focal infection, yet in the absence of his previous insurance history and disregarding focal infection as a possible cause of trouble, he would, on the basis of his examination of July 31st, be considered a standard risk. The symptoms while important, were transient and might readily have escaped detection, whereas the presence of an objective efficient cause, being persistent, would be readily ascertained on examination.

A second case, E. H. E., age 30, wholesale merchant, applied for insurance but was declined in June, 1922, owing to the presence of a fairly marked albuminuria. During July and August although under treatment the albuminuria was found at frequent intervals, recurring especially after exercise, as playing golf. He was easily tired, did not look robust and had suffered from palpitation occasionally for the past 6 months. The family history was good; he had suffered from no previous illness and his habits and manner of living were satisfactory.

He consulted me Sept. 2, 1922. Examination showed marked oral and tonsillar infections and there were 6 dead teeth—3 devitalized crowned teeth and 3 pivots; the gums in the areas of the devitalized teeth were swollen and purplish-red in color. The tonsils were small but firm, adherent, cryptic and contained a great deal of caseous debris; marked redness and hypersecretion of the throat.

The urine showed a distinct trace of albumin with a few hyaline casts and some cylindroids. Renal function tests were satisfactory; Wa. R. negative. No other evidence of disease was found. After consultation with his dentists, the three crowned teeth, all showing marked periapical infection, were extracted. He began to improve rapidly. There is still a faint trace of albumin but his foci of infection have not been entirely eradicated. From repeated experience with this type of case I have little doubt that ridding him of his focal infections will result in his albuminuria disappearing and his becoming acceptable as a standard risk. Otherwise his symptoms might clear at intervals for a time but I do not

think he would ever be considered as a standard case so long as such a severe grade of focal infection remained.

This case appears to me to represent a type and degree of focal infection in which a rating should be imposed, even had albuminuria not been discovered.

Dr. McMahon—I will call on Dr. Fraser to lead the discussion of this paper.

Dr. Fraser—It seems to have been rather the order of the day to apologize for having come to discuss certain subjects and so you may consider that my apology is in also.

I feel very much in the position of the old darkey minister who had been sent down to a very unregenerate town in the South and after working hard in his evangelizing capacity he had been unable to turn anybody towards repentance. At last he was heard to pray, "Oh Lord, thou knowest I has been workin' hard; thou knowest I has done my best; but Oh Lord, I have done no good; Oh Lord, I need help; come down and help me; come down yourself; don't send your son; dis am no place for a boy."

Now I feel that this is no place for a boy, and the man to discuss this question of Focal Infections is my chief, Dr. Rogers, because this subject has always been pie for him.

It has been a great pleasure to me to look over a number of cases with Dr. Anderson on my visits to Toronto, and to discuss with him the question of Focal Infections. I knew that he was very enthusiastic in this matter, and when I heard that he was going to prepare a paper, I felt it was about time to get a knife out, as I didn't quite accept all the enthusiasm. But when I read his paper, I was delighted with the conservatism expressed in his ideas, and with the questions that he has raised in connection with Focal Infections. It is unfortunate at the present time that we are passing through one of those transitions in medicine whereby everybody suddenly siezes on a new idea, chews it up, spits it out again, all before they have found out whether it was meat to eat or not. Not very long

ago, when we met a person on the street, he would proudly reach into his pocket and pull out and exhibit with great pride the appendix which had been taken out; but now, when we meet our friends on the street, with their lips twisted and their jaws distorted, and mumbling like old grandmothers, they proudly reach into their pockets and show us pictures from the rentgenologist, of all sorts of teeth, they will show us where the ink marks have been put to indicate that they have had abscessed cavities and that their teeth must come out, and many a good tooth has been pulled out by reason of those pictures. I am very conservative with regard to these Focal Infections, just at the present time. I want to wait a little before I accept as a fact that all the disorders that the human flesh is heir to are due to his teeth and due to his tonsils. There are a lot of other organs that can show infection, but they haven't yet advised that they all come out. Only a short time ago a report was published by some Frenchmen in connection with tubercular cases in which they showed that by taking out the appendix all of the signs in the lungs had cleared up. Most of us have lost our appendices, and we don't need to consider the cure of tuberculosis.

The nubbin of Dr. Anderson's paper appears in the last paragraph in which he says the question naturally arises what one's attitude should be in a case where the foci of infection in the teeth and tonsils supposed to be the cause of certain symptoms, have not been entirely eradicated. I dare say that every one of you have during the last few months been reading the papers on your desk, and finding a long record of albumin and casts and sugar, or of heart murmurs or functional heart trouble, and you have read that this applicant has had his tonsils taken out some months before, and now the arrhythmia has departed, the albumin and the casts have disappeared, or he has had his teeth X-rayed and taken out, and now all the damages have been removed. The question then arises, can we take those cases even supposing the conditions have cleared up, on a standard basis? Can we take cases that have been subject to a prolonged and assidu-

ous poison on the same basis as a person who has never been poisoned under those conditions? I think that is the nubbin of the whole thing. We have no data at the present time, except a few cases that have shown apparent cure because the teeth or the tonsils have been taken out. I think we ought to go very cautiously before we accept the fact that Focal Infections cause all the damage that we see marked down in our records.

Dr. Gordon Wilson—I think the question of the proof of the theory of Focal Infections in their bearing on metastatic conditions is really founded on statistical studies; namely, first, the report of the British Medical Association; and, second, the work of Lambert, of New York, in showing the decreased number of cardiac cases admitted to Bellevue since tonsillectomies had become frequent.

There is one point I think that all of us should remember and that is, that pyorrhœa often plays probably a bigger part in these infections than apical abscesses. In 1875, Dr. J. W. Riggs, dentist, of Hartford, Conn., wrote an article on this subject in which he showed a case of iritis and rheumatism which had been practically cured by the removal of some teeth and curing of the pyorrhœa, and the interesting fact on that paper was the statement he made, namely, that instead of saying that bad teeth were the result of old age, a more correct interpretation would be that signs of old age are probably the result of bad teeth.

There has been lately made, however, a very thorough study of the bacteriology of these cases of apical abscess, and there is an excellent report in the April number of the National Dental Association, which is of interest. It reports on cases studied by the Research Committee—cases of myocarditis and associated diseases which we generally look upon as being in a measure due to Focal Infections, and that report brings out that in the study of these with reference to the teeth, that 33% were absolutely negative on culture.

There is a point which Dr. Anderson has brought out and which I think is of especial interest and that is, that the physi-

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cian is a far better judge than the dentist of these Focal Infections. Personally, I believe we have got to consider the question of a more favorable consideration in those applying for insurance who have had removed not only the foci of infection, the teeth, but also the *portals* of infection, the tonsils. If the tonsils have lost the power of destroying germs then they should be removed, and the general feeling to-day is, that you practically never make a mistake in removing questionable tonsils in those under 20, but in those over 30, the tonsils must be undoubtedly the cause of disease to justify their removal.

What I would particularly emphasize in Dr. Anderson's paper is the fact that the medical man and not the dentist is the best judge of teeth and gums in relation to general health, and therefore our examiners can give us information of value.

Dr. Exton—I hate to see a good paper like this go with so little discussion, not only because the matter is very important, but also because the question which has been raised by Dr. Anderson goes to the very heart of it.

In the light of the available evidence there is no question but that albuminuria and other conditions happen as the result of focal infections, and the work which has been done by Professor Rosenow and his co-workers at the Mayo Clinic with selective strains of bacteria is to my mind absolutely convincing.

As Dr. Anderson says: The important point of interest to Medical Directors is, what may we expect after a focus has been discovered and has been removed and the process has apparently disappeared? Will these people remain cured or are they likely to have recurrences?

From what I can gather, opinion seems pretty well crystallized to the effect that if the focus is one which can be satisfactorily and thoroughly drained and has been treated in such a way as to clear up perfectly and remains clear for an appreciable time in such cases it is reasonable to regard the condition as permanently cured. If, on the other hand, the focus is in one of the sinuses, or in the digestive tract, or in

some inaccessible location where perfect drainage cannot be surgically contrived, or if the case is one in which treatment has not been radical enough, in such cases recurrences may reasonably be expected.

Dr. Hobbs—Mr. President: This discussion brings to mind a case that I have knowledge of, a case of well-marked nephritis, in which the gentleman had had his teeth out, the albumin casts had disappeared, and everybody was happy. It was not a case for insurance, but one which I happened to know personally. In two years the man died of nephritis and yet everything had been reported as cleared up, and his family were very happy over the result that followed immediately the extraction of his teeth. Perhaps a longer period would tell a little more than a shorter period in these cases.

Dr. Anderson—Mr. President: I entirely agree with what Dr. Fraser has said with reference to extreme views in this matter of Focal Infections. It is necessary to exercise good sense and discrimination the same as in any other matter. In connection with the material forming the basis of my paper we kept a very careful record over a number of years of all cases of oral and tonsillar infection and the impairments associated therewith—over 2,000 cases in all.

The question whether when the infection has been removed the case can be accepted as standard or not, I think would have to be decided in each case on its own merits. Dr. Hobbs has referred to the same question. If an infection has existed for a length of time and organic disease has resulted from it, certainly the removal of the original cause does not necessarily get rid of the result, and I think it is very important to remember this. On the other hand, when we consider this question of Focal Infection of the teeth, what it all means, it is necessary to visualize a sealed-in streptococcic infection at the apices of from 80 to 100% of all devitalized teeth. This should impress anyone that we are dealing with a very important matter, especially in cases where one would find a dozen or more devitalized crown, pivot or stopped teeth. Unnecessary extraction must be deprecated as strongly as possible.

It is not a matter of getting rid of the teeth—it is a matter of getting rid of a serious form of infection in which there is no proper drainage.

As to whether drainage of the focus removes the trouble, there again we have to remember that in case of dead teeth, they usually have died as the result of infection and if the pulp is destroyed, the chief part of the nutrition of the teeth is cut off. Now you will get a certain amount of nourishment from the peri-dental membrane, but often it is coincidentally involved by pyorrhoea. Draining such teeth and still leaving them there, you may relieve symptoms for a time, but the symptoms will certainly recur. Just as if you left an infected foreign body under the skin, you might open the abscess, drain it, apply iodine to such a foreign body and close up the opening, but from the pathology of the condition, we know that the infection would not be removed and the symptoms would recur.

I believe myself, and this belief is confirmed by the examination and history of the cases which I have followed, that in many instances where we get reports extending over a period of ten or more years, of a number of slight, often dissimilar ailments, returning from time to time such as albuminuria, then a period of freedom, then a history of irregular heart action or a heart murmur, then a recurrence of albuminuria, etc., we have some underlying cause for these symptoms which has not been removed and which tends to make the condition progressive. Examining such a case at a period when the impairment does not show up, does not mean that, if the cause remains, it is a safe case for insurance at ordinary rates: I am satisfied that the underlying cause in many cases is one of these persistent focal infections: and that we ought to pay more attention to that factor in the selection of risks.

Dr. McMahon—A Meeting of the Association of Life Insurance Medical Directors would not be complete without a contribution from Dr. Rogers and Mr. Hunter, and they will now present to us a Mortality Study of Impaired Lives.

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MORTALITY STUDY OF IMPAIRED LIVES

A. INFLAMMATORY RHEUMATISM.

B. TUBERCULOSIS OF LUNGS AND BLOOD SPITTING.

BY OSCAR H. ROGERS, M.D.

Chief Medical Director

AND

MR. ARTHUR HUNTER, F. A. S.

*Chief Actuary of the New York Life Insurance Company
of New York, N. Y.*

In a paper which we read before this Society in October last, we discussed the insurance significance of certain medical impairments, and wish now to add the results of our studies of certain other of them, a history of inflammatory rheumatism, mitral regurgitation in connection with a history of inflammatory rheumatism, a history of blood spitting, and of old lung trouble or cured tuberculosis.

All of the risks contained in these groups were insured by the New York Life on underaverage plans, during the years 1896 to 1920 inclusive, and studied up to the policy anniversaries in 1921. The study is by policies and not by lives nor by amounts insured. During the years 1896 to 1906 inclusive, these policies were issued in a special class as to dividends and those risks that were thought to be more seriously impaired had also a lien placed against them. From 1907 to 1920 inclusive, they were issued with an advance in age.

There is no table of mortality in existence which represents the experience of this Company on standard lives during the entire period covered by this investigation. In the earlier years of that period the Company's general mortality was represented by the Compound Progressive Table, in the later years by the Medico-Actuarial Table and in the intermediate period somewhere between these two tables. In order that the mortality ratios published in this paper may be compared with those of other companies engaged in similar investigations, we

have employed the Medico-Actuarial Select Table. At the same time, in order to give a more accurate expression of the mortality in the classes under discussion compared with the Company's general experience on standard lives, we have also used as a standard a table midway between the Compound Progressive and the Medico-Actuarial. The ratios thus derived appear under the heading, "Ratio of Actual to Expected Deaths by the Company's Experience on Standard Risks." It will be understood, of course, that these ratios are approximate only.

During the period under consideration, the standards employed by the Company in placing valuations upon these lives have changed from time to time, according to the information we have been able to secure, so that the selection has varied somewhat in severity. We may also say that, in order to increase as much as possible, consistent with reasonable homogeneity, the material contained in these classes, we have included a few minor impairments. We are satisfied however, after a careful review of the records, that the results of this study cannot have been modified as much as 10% of the M. A. Table by that procedure, and we believe that it is highly probable that it has not been influenced by more than 5% by their inclusion. When interpreting the results of this investigation, all of these facts should be taken into account.

History of Inflammatory Rheumatism

The material, 5876 cases, was divided according to the number of attacks and also to the time elapsed since the last attack as follows:

There were three other groups in which the experience was not sufficiently large to justify publication,—“One attack at some time in the past, not specified,” “Two or more attacks within 3 to 5 years prior to application,” and “Two or more attacks 6 or more years prior to application.” The aggregate experience in these groups, probably on account of accidental fluctuation, was somewhat better than any of the four groups submitted.

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These results do not appear to be entirely consistent, as they show a higher mortality among persons with one attack than

	Number of Cases	Actual Deaths	Expected Deaths by M. A. Table	Ratio of Actual to Expected	
				By M. A. Table	By Co.'s Experience on Standard Risks
One attack within 2 years.....	1276	84	52.5	160%	145%
One attack within 3 to 5 years.....	766	63	38.6	163	150
One attack more than 5 years ago.....	1667	178	122.9	145	135
Two or more attacks within 2 years.....	1174	112	75.2	149	140

with two or more attacks, the last one within two years prior to the date of application. This may possibly be due to accidental fluctuation or to the more careful selection practiced in the latter cases.

Combining the data for the four groups the following are the results for ages at entry 15 to 39 and at 40 and older.

Ages at Entry	Actual Deaths	Expected Deaths by M. A. Table	Ratio of Actual to Expected	
			By M. A. Table	By Co.'s Experience on Standard Risks
15-39.....	197	120.8	163%	145%
40 & over.....	240	168.5	142	135

An analysis of the four groups showed that in two of them the relative mortality was lower at the younger than at the older ages, while in the other two the reverse was the case. Clearly this experience does not justify us in making in our ratings any differences based upon ages at entry.

The deaths from heart disease were 20% of the total deaths, a high rate compared with that among standard risks.

In drawing deductions from this experience it should be remembered that applicants with a record of an attack of inflammatory rheumatism of ordinary severity were usually given standard policies and are, therefore, not to be found in the data now presented. The attack of rheumatism must have been sufficiently severe to call for a rating in order to bring them within the group we are now studying. Our experience therefore shows the effect on mortality of the more severe attacks of inflammatory rheumatism and should not be compared with the experience with carefully selected standard lives to which standard insurance had been granted.

The ratings which we employ at the present time are as follows:

	ACUTE ARTICULAR RHEUMATISM		
	Within 1 Yr.	1 to 5 Yrs.	Over 5 Yrs.
One attack.....	+30	+20	+10
Two or more attacks.....	+40	+25	+15

While our standards have varied during the twenty-five years covered by this investigation, we have constantly adhered to the policy of decreasing the rating with the lapse of time since the attack. The more recent views of the medical profession regarding the causation of rheumatism and the treatment of it by the elimination of the causes of infection, leave us satisfied that we have done this. Acute articular rheumatism is now looked upon, and is being treated as, an infectious disease and recurrences of it are being avoided by the removal of foci of infection, usually in the tonsils, the teeth or the nasal sinuses. Even with this improved mastery over the disease, we believe that the testimony is sufficient to justify insurance companies in looking upon a severe attack of acute articular rheumatism during the years immediately after the

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attack as a distinct impairment. In our ratings we have taken account of the increasing knowledge of rheumatism in that our standards are more favorable than our experience.

Rheumatism and Mitral Regurgitation

In our paper published in 1919 on "Heart Murmurs, Their Influence on Longevity," we gave the experience of the New York Life Insurance Company in a group of cases of mitral regurgitation, associated with a history of inflammatory rheumatism. The mortality in this group was very high, a result so unexpected that we have made a further investigation of our material in order to determine whether or not there has been any change in the last three years.

Our report of 1919 covered 1868 cases insured from 1896 to 1917 both inclusive. The present study represents that old material plus such cases as have accumulated down to 1920 inclusive, carried to the policy anniversaries in 1921. As in our former study, we have divided the cases into two groups, (a) those with little or no hypertrophy, (b) those with moderate hypertrophy. Cases with a substantial amount of hypertrophy have been declined altogether. As in the preceding group, the attack of rheumatism was in all cases sufficiently severe and recent to call for a rating.

Mitral Regurgitation with little or no Hypertrophy with History of Inflammatory Rheumatism

	No. of Cases	Actual Deaths	Expected Deaths by M. A. Table	Ratio of Actual to Expected	
				By M. A. Table	By Co.'s Experience on Standard Risks
One attack.....	1426	169	64.1	263%	240%
Two or more attacks.....	612	106	30.3	350	320
Total.....	2038	275	94.4	291	270

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With regard to the cases in which there was a moderate degree of hypertrophy, the material, though of small volume, is given subdivided by number of attacks of inflammatory rheumatism.

Mitral Regurgitation with Moderate Hypertrophy with History of Inflammatory Rheumatism

	No. of Cases	Actual Deaths	Expected Deaths by M. A. Table	Ratio of Actual to Expected	
				By M. A. Table	By Co.'s Experience on Standard Risks
One attack.....	346	51	14.9	343%	315%
Two or more attacks.....	154	35	6.8	515	470
Total.....	500	86	21.7	396	360

This study strengthens the conclusion drawn from our previous experience that these cases of heart murmur with a moderate degree of hypertrophy call for a high rating and that those with considerable hypertrophy are practically uninsurable. The hazard involved in "Mitral regurgitation with a history of acute articular rheumatism" is much greater than the sum of the separate ratings. Those who employ the numerical method of valuation should make due provision for this.

The percentage of deaths from organic heart disease was very similar to the previous experience. Of the total deaths in the group of mitral regurgitation with little or no hypertrophy, 56% were from heart disease and in the group of those with a moderate degree of hypertrophy 62%.

A comparison of the age distribution shows that 20% of these cases were among applicants 40 years of age or over, while among the group of those with a history of acute articular rheumatism without a heart murmur at the time of application, the corresponding ratio was 33%.

We may add, by way of a side light upon this subject, that

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in an analysis of 800 cases of mitral regurgitation a history of acute articular rheumatism was disclosed in 14%. In making this analysis we took an equal number of cases with and without hypertrophy. There was no practical difference in the percentage of cases with acute articular rheumatism in these two groups. Of the 14%, 4% received an additional rating because of rheumatism and 10% received no additional rating. In the cases which were not rated the last attack occurred a number of years before the date of application,—on the average about fifteen years.

The company's standard ratings for mitral regurgitation without a history of rheumatism are as follows:

	Very Best No Hyp.	Average No Hyp.	With Slight Hyp.	With Moderate Hyp.	With Considerable Hyp.
Mitral Regurgitation..	+60	+75	+100	+125	declined

Old Lung Trouble, Cured Phthisis, Hemoptysis and the Like.

One of the most interesting questions with which we have to deal is the effect upon longevity of consumption or tubercular disease of the lungs. Fragmentary studies of our own, the first of them in 1900, early satisfied us that the relative mortality in these cases is much heavier among the light weights than among those of heavy build; also heavier among young persons than among older lives. These facts are shown in class 55 of the Specialized Mortality Investigation, especially if we apply to the ratios there published the correction necessary to make them express the mortality compared with the true mortality of the contributing companies during the period covered by the investigation. Again, classes 13, 14 and 23 of the Medico-Actuarial give us a still broader view of the subject. Both of these investigations had to do with standard lives, with which the present material may not properly be compared.

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This is a study of lives of this sort which have been insured on substandard plans in the manner already described. With regard to applicants for insurance who have had tuberculosis or been suspected of having it either because of a suggestive history or of physical signs, it has been the practice of the Company to keep record of three different types of impairments:

A. Cases presenting abnormal physical signs, such as dullness, suspicious apices, rales, area of consolidation and the like.

B. Cases giving a history of blood spitting or hemoptysis, but without physical signs or definite history of disease.

C. Cases that have frankly suffered from tuberculosis of the lungs and recovered from it.

It was thought that the majority of these cases would fall in the last of these categories, group C, or in it in combination with either A or B. That is to say, we should expect a history of old lung trouble with blood spitting or with physical signs of the disease found on examination. It was found, however, that in only 35% of our cases was this true; that in 40% of them there was a history of blood spitting alone and in 25% some physical signs without any record of actual disease. This result was so surprising that we reviewed the original papers with great care in a number of cases chosen at random to see if our card records were correct. We found no evidence of error, but did find that in some of the cases of hemoptysis or blood spitting there was a record of "a rundown condition" at some time in the past. We also found in some of the cases in group A that the applicants had no idea of their condition and in other cases there was a history of only pleurisy, pneumonia or an attack of bronchitis.

We have studied this material in three groups:

(a) Physical signs in lungs on examination without a history of tuberculosis.

(b) A history of blood spitting without a definite history of tuberculosis.

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(c) Cured tuberculosis of the lungs with or without either a history of blood spitting or physical signs on examination.

This material was divided into three weight groups as follows:

- (1) Underweight—persons 10% and more underweight.
- (2) Average weight—persons from 9% underweight to 9% overweight both inclusive.
- (3) Overweight—persons 10% and more overweight.

A study of the material in group (a) divided into the three weight groups gave the following:

	No. of Cases	Actual Deaths	Expected Deaths by M. A. Table	Ratio of Actual to Expected	
				By M. A. Table	By Co.'s Experience on Standard Risks
Underweight...	498	49	23.9	205%	190%
Average weight	594	47	26.0	181	165
Overweight.....	173	14	8.3	169	155

These results are in accordance with the expectation that the mortality should be greatest among the underweights.

A study of the material in group (b) persons giving a history of blood spitting without a definite record of tuberculosis, showed the following:

HISTORY OF BLOOD SPITTING

	No. of Cases	Actual Deaths	Expected Deaths by M. A. Table	Ratio of Actual to Expected	
				By M. A. Table	By Co.'s Experience on Standard Risks
Underweight...	752	195	55.1	172%	160%
Average weight	1219	13	69.1	164	150
Overweight.....	307	25	19.3	130	120

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Here again we see the higher mortality of the underweights shading down to the comparatively low mortality among the overweights, but it is to be noticed that the mortality in this group is not as high as it was found to be in group (a).

In the last group (c), we included all cases with a history of pulmonary tuberculosis, whether with physical signs or a history of blood spitting or with neither of them.

HISTORY OF CURED PHTHISIS

	No. of Cases	Actual Deaths	Expected Deaths by M. A. Table	Ratio of Actual to Expected	
				By M. A. Table	By Co.'s Experience on Standard Risks
Underweight...	569	41	24.6	166%	155%
Average weight	1045	71	34.1	208	190
Overweight.....	292	15	9.8	153	145

This experience is notable in that the mortality among the underweights is better than that among persons of average weight. Naturally the company has been very careful in its selection of light weights with a history of tuberculosis and the comparatively low mortality among those underweights may be due to the extremely rigorous selection practiced among them.

In order to get an expression of both the influence of age and of the time elapsed since recovery from the disease, groups (b) and (c) were combined and subdivided according to age and according to whether the attack was within five years or more than five years of the date of application. A subdivision by weight groups of persons aged at entry 40 and over whose last attack occurred within 5 years prior to application, is not given, as there were only 2 deaths in the underweight and 5 in the overweight group. The following table gives a synopsis of the result:

This table brings out well the fact that the mortality is exceedingly heavy among young persons only recently recovered from the disease; also that it is comparatively heavy

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	Actual Deaths	Expected Deaths by M. A. Table	Ratio of Actual to Expected	
			By M. A. Table	By Co.'s Experience on Standard Risks
<i>Last attack within 5 years</i>				
<i>Ages 15-39</i>				
Underweight.....	45	13.6	330%	295%
Average weight..	73	27.9	262	235
Overweight.....	11	5.8	188	170
<i>Age 40 and over</i>				
	17	16.1	106	100
<i>Last attack over 5 years ago</i>				
<i>Ages 15-39</i>				
Underweight.....	45	26.6	169	155
Average weight..	60	36.4	165	150
Overweight.....	10	9.1	110	100
<i>Age 40 and over</i>				
Underweight.....	44	35.7	123	115
Average weight..	41	31.6	130	125
Overweight.....	14	9.0	155	150

among light weight and medium weight applicants under 40 years of age, even though the last attack occurred more than five years ago. It is interesting to note too that each group shows a mortality substantially higher than normal, excepting where the amount of material is so small as to raise a question of accidental fluctuation. In the overweight group aged 40 and over last attack over 5 years ago, the fact of overweight may be more significant than the history.

A review of the causes of death in these three groups shows that 35% of the deaths in group (a) was due to tuberculosis, 35% in group (b) and 38% in group (c)—a uniformly high mortality from tuberculosis of the lungs in all three groups. Combining all of the groups and studying them from the standpoint of build, the deaths from tuberculosis of the lungs stood at 42% among underweights, 36% among persons of average weight and only 13% among those in the overweight group.

Influence of Climate

It is often claimed by those who live in climates or parts of the country specially famed for their salubrity, that tuberculosis among them is a much less formidable disease than it is elsewhere, and that a tubercular process arrested there rarely gives trouble afterwards. To test this out, we set apart the risks in this study which at the time of their acceptance were residing in the eight mountain states, Idaho and Montana on the north to New Mexico and Arizona on the south. There were 264 of these lives with 28 deaths among them, 11 deaths expected by the M. A. Table, a ratio of 255%. Of the 28 deaths, 15 were from tubercular disease. While the amount of material is too small to justify any very positive conclusion, it is large enough to suggest that the mortality among persons of this sort living in the so-called mountain states at time of application is apparently not better than among those living in other states at time of application.

The following is the table of numerical ratings which we now employ:

HISTORY OF TUBERCULOSIS OF LUNGS OR OF BLOOD SPITTING*A—Without Physical Signs of Old Lung Trouble*

Age at Entry	No. of years elapsed since attack ¹	Percentage under or over weight				
		-20%	-10%	0	+10%	+20%
25	2-5 yrs.	+260	+175	+125	+80	+60
	5-10 "	+170	+115	+80	+55	+40
	over 10 "	+80	+55	+40	+25	+20
35	2-5 "	+190	+130	+90	+65	+40
	5-10 "	+125	+80	+60	+40	+30
	over 10 "	+60	+40	+30	+20	+15
45	2-5 "	+95	+65	+45	+30	+20
	5-10 "	+60	+40	+30	+20	+10
	over 10 "	+30	+20	+15	+10	+5

¹ We do not accept cases where the applicant gives a history of tuberculosis of lungs or blood spitting with the last attack within two years of application.

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B—With Physical Signs of Old Lung Trouble

25	2- 5 yrs.	+370	+260	+175	+125	+80
	5-10 "	+235	+170	+115	+ 80	+55
	over 10 "	+130	+ 85	+ 60	+ 40	+30
35	2- 5 "	+280	+190	+130	+ 90	+60
	5-10 "	+170	+125	+ 80	+ 60	+40
	over 10 "	+100	+ 65	+ 45	+ 30	+20
45	2- 5 "	+145	+100	+ 65	+ 40	+30
	5-10 "	+ 85	+ 60	+ 40	+ 30	+20
	over 10 "	+ 50	+ 35	+ 25	+ 20	+10

In the practical application of these ratios to current business we apply them to tubercular disease of the lungs as a whole, including a simple history of hemoptysis, not clearly traumatic, or lung signs without any history, on the one hand, and a history of consumption with old lung signs, on the other. As in all numerical ratios these are central values and the ratings in individual cases should be shaded from these according to the merits of the case. This is where a discriminating medical judgment is most useful.

Dr. McMahon—In this connection before discussing this paper of Dr. Rogers and Mr. Hunter, we will have presented to us a study by Dr. Rockwell on the experience of his Company on risks with Mitral Regurgitation.

EXPERIENCE ON RISKS WITH MITRAL REGURGITATION. ISSUES 1900 TO 1918

OBSERVED TO ANNIVERSARIES IN 1919

BY DR. T. H. ROCKWELL

Medical Director, Equitable Life Assurance Society

At the last meeting of the Association, I explained the methods we are now using in the selection of impaired hearts and the rating accorded them.

Up to 1900 all cases of cardiac impairment were declined.

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In that year we commenced to write policies on impaired lives. There was very little to guide us for no competent data received up to that time was available. We felt by limiting the age of entry to forty on the Endowment plan, with a slight extra premium, we could safeguard ourselves, believing that the mortality would be a deferred rather than an immediate one. So we provided on the average for an extra mortality of plus 40 or 45 for a mitral regurgitant murmur described as being "well compensated." It was not long before we began to pay death claims and discovered that the extra mortality began soon after the issuance of the policy and was not deferred to the point we hoped it would be. This is particularly shown in the present experience in the two first policy years. As soon as we had enough experience to justify it, a study was made of our heart class, with the result that we more than doubled the extra ratings. For the last ten years these ratings have been in use with of course slight variations made at times in conformity with the numerical system we employ. They are as follows:

	Very Best No Hyper- trophy	Average No Hyp.	With Slight Hyp.	With Moderate Hyp.	With Consider- able Hyp.
Mitral Regurgitation....	+60	+75	+100	+125	D
Aortic Obstruction.....	+75	+100	+125	+150	D
Aortic Regurgitation....	+200	D	D	D	D
Mitral Obstruction.....	+200	D	D	D	D

From the experience I now present to you it would seem that our present method of selection of cases of Mitral Regurgitant Murmur—with or without hypertrophy—and with or without a history of rheumatism, should protect the interests of the company, and at the same time give the benefits of insurance to many who have heretofore been denied it.

The experience tabulated below has been prepared on the basis of policies. In the earlier years of our substandard business, policies were issued for much larger amounts than are

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permitted under our present rules; there are, accordingly, wide variations in the amounts of individual insurance under the policies covered by this experience so that, in view of the paucity of the data, it is felt to be hardly worth while to present the experience by amounts of insurance.

In order to show the effect on the experience of the War and the Influenza Epidemic of 1918 and 1919, the tabulations also show the corresponding numbers of policy claims due to these causes, and the ratio of the numbers of the actual claims to the expected.

I. Without Hypertrophy or other *Important* Impairments.

<i>Experience by Policies</i>					
Entrants	Actual Claims	Ratio of Actual to Expected by Equitable S. & U.	War Claims	Epidemic Claims	Ratio of Balance of Claims to Expected
2208	119	159%	3	3	151%

II. With Hypertrophy but without other important impairments.

1952	72	217%	1	10	184%
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In the earlier years of our substandard business policies were issued for larger amounts than subsequently. There were four claims aggregating \$150,000 in Classes I and II, which materially increased the percentage of amount over the mortality by policies. At the present time our limit during the first year is not more than \$15,000 on the average.

III. With and without Hypertrophy and with a history of Rheumatism:

<i>Experience by Policies</i>					
Entrants	Actual Claims	Ratio of Actual to Expected by Equitable S. & U.	War Claims	Epidemic Claims	Ratio of Balance of Claims to Expected
405	34	429%	1	2	391%
(By lives the experience was 381)					

IV. With and without Hypertrophy and with miscellaneous other Important Impairments:

1130	49	155%	—	4	142%
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There are 5695 entrants (policies) in the above 4 groups of risks with mitral regurgitation. 239 of these substandard policies were subsequently changed to standard. From the time of their change they are eliminated from the above mortality experience.

Group IV, comprising risks with mitral regurgitation, with or without hypertrophy, and with miscellaneous other important impairments, shows a mortality no larger than Group I. The reason for this might be in the smallness of the group, or it might be that generally where another important impairment was found, the mitral regurgitation or the other important impairment was of a below average severity.

The risks of Group IV which became death losses show below what were classified as "other important impairments":

- | | |
|--|---|
| 1. Systolic murmur at apex. Some Hypertrophy. Forcible Heart Action. | Wholesale liquor dealer. |
| 2. Mitral Regurgitation with full compensation. | Severe nervousness for three months two years ago. |
| 3. Systolic murmur at apex. | Hemorrhoids or ulcers 4 months ago. Operation. Otorrhea since childhood. Rheumatism 1 month 7 yrs. ago. |
| 4. Mitral insuff. | Hotelkeeper. |
| 5. Very small mitral systolic murmur. | Heavy, 5-11-220. Waiter in saloon. Slight rheumatism. Pain in right knee for several years. |
| 6. Mild Mitral Regurgitation. | Albumin, Casts, inconstant. |
| 7. Very slight murmur at apex. | Pulse 78-somewhat irregular. Pulse 96-Regular. |
| 8. Mitral Regurgitation. | Small amount of albumin, hyaline casts. |

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|--|---|
| 9. Mitral Regurgitant Murmur. | Underweight. Some rheumatism 6 years ago; no other details. |
| 10. Mitral Regurgitation, some enlargement. | N. Y. City Fire Dept. |
| 11. Mitral Regurgitation. | Collector Brewery. |
| 12. Mitral Regurgitation some enlargement. | Irregular Pulse. |
| 13. Mitral Regurgitation some enlargement. | Blood pressure 140. |
| 14. Inconstant mitral regurgitation. | Light weight 5-10-125, age 21. |
| 15. Mitral Regurgitation, some Hypertrophy. | Action irregular, intermittent. |
| 16. Mitral Regurgitation Atheroma. | Albumin Hyaline Casts. |
| 17. Systolic murmur at apex. | Occ. |
| 18. Mitral Regurgitation moderate enlargement. | Pulse 100. |
| 19. Inconstant syst. murmur at apex. | Walls of arteries slightly thickened. |
| 20. Dr. A. Mitral Reg. Murmur full compensation.
Dr. D. moderate enlargement no murmur. | Overweight. |
| 21. Mitral Regurgitation. | Sugar 0.42 inconstant. Infl. Rheum. at age 17. |
| 22. Mitral Regurgitation heart enlarged. | Pulse 90. |
| 23. Mitral Regurgitation some hypertrophy. | Pulse 90+. |
| 24. Mitral Regurgitation compensated. | Blood Pressure 150. |
| 25. Mitral Regurgitation slight enlargement. | Blood Pressure 155. |

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|---|--|
| 26. Mild Mitral Regurgitation. | Abortion last March. |
| 27. Very slight Mitral Regurgitation. | Intermittent Pulse. |
| 28. Mitral Systolic Murmur. | Irregular heart action. |
| 29. Mitral Systolic Murmur only after undue exercise. | Irregular pulse. |
| 30. Mitral Regurgitation. | Pulse 60—irregular. |
| 31. Mild Mitral Regurgitation. | Intermittent Action. |
| 32. Slight Mitral Regurgitation. | Pulse intermittent twice a minute. |
| 33. Faint syst. murmur at Apex. | Overweight. |
| 34. Mitral Regurgitant murmur. | Blood Pressure 140—Heavy. |
| 35. Mitral Reg. | Overweight. |
| 36. Mitral Insuff. | Hotel Prop. Tends bar. |
| 37. Mitral Reg. Murmur. | Albumin. |
| 38. Mitral Reg. Murmur heart enlarged. | Albumin small amount. |
| 39. Mitral syst. murmur. | Trace of albumin, hyal. casts. |
| 40. Mitral syst. murmur some enlargement. | Trace of albumin, hyal. casts. |
| 41. Mitral Reg. murmur. | Heart very irregular. |
| 42. Mitral Reg. murmur enlarged. | Action intermittent at long intervals. Overweight. |
| 43. Mitral Regurgitant Murmur. | Lightweight. |
| 44. Mitral Regurgitant Murmur. | Lightweight. |
| 45. Mitral Regurgitant Murmur. | Pale anæmic looking. |

Discussion—Impaired Lives

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History of Rheumatism

We have on record 560 substandard policies with a history of rheumatism as the only impairment. The experience from 1900 to 1919 under these risks was as follows:

Entrants	Actual Claims	Ratio of Actual to Expected by Equitable S. & U.	War Claims	Epidemic Claims	Ratio of Balance of Claims to Expected
560	29	134%	1	2	120%

Ratings for Rheumatism

Acute Articular (Exclude Muscular Type)

	Within 1 year	1 to 5 years	After 5 years
1 attack.....	+30	+20	+10
2 or more attacks.....	+40	+25	+15
Muscular, unless very severe or frequent and if diagnosis from articular rheumatism is certain.....			+0

In the groups where Mitral Regurgitation is the principal impairment the deaths from circulatory diseases are about twice as numerous as they are among standard risks.

Dr. McMahon—Dr. Russell will open the discussion.

Dr. Russell—The results of the Mutual Life Insurance Company in regard to acute articular rheumatism, may be of interest to the Association. The mortality experience is considerably lower in this group of cases than that of the New York Life, as illustrated in the following table:

One Attack within 5 Years

Ages	Entrants	Exposures	Deaths		Ratio
			Actual	Expected	
15-39	1012	3904	17	18.07	94.1
40 & over	264	1158	8	12.29	65.1
Total.....	1276	5062	25	30.36	82.4

2 or more Attacks within 5 Years

15-39	151	572	3	2.69	111.7
40 & over	60	248	5	2.92	171.3
Total.....	211	820	8	5.61	142.6

1 or more Attacks within 6-10 Years

15-39	693	1754	5	7.81	40.
40 & Over	530	1327	13	13.82	94.1
Total.....	1223	3081	18	21.63	83.3

Total 1 or more Attacks

15-39	1856	6230	25	28.57	87.5
40 & Over	854	2733	26	29.03	89.6
Total.....	2710	8963	51	57.60	88.5

Issues of 1907 to 1917 exposed to 1918—Mutual Life experience of cases with a history of Acute Articular Rheumatism by the Medico-Actuarial Select Table.

The total result in cases of one or more attacks, ages at entry 15 to 39, was 87.5% as compared with 163% of the New York Life. The Mutual experience for ages 40 and over was 89.6% as compared with 142% of the New York Life. The Company's general experience for the younger ages was 73.7, and in the older group 76.1. The causes of death are as follows:

ISSUES OF 1907-1917—EXPOSED TO 1918
(ACUTE INFLAMMATORY RHEUMATISM)

Deaths from all causes.....	51	Heart Disease.....	7
Typhoid.....	7	Angina Pectoris.....	2
Influenza.....	2	Arteriosclerosis.....	1
" Epidemic.....	2	Pneumonia.....	5
Tuberculosis.....	3	Dis. of Liver & Gall Bladder..	1
Cancer.....	3	Appendicitis.....	3
Apoplexy & Paralysis.....	3	Bright's Disease.....	1
		All others.....	11

Heart Disease accounts for 14% of the deaths as against 20% of the New York Life. Acute infectious diseases accounted for 23% of the deaths. This high mortality from acute infectious diseases, where a previous history of rheumatism is given, is probably due to the fact that a damaged heart already existed, which could not stand infection.

Dr. Rogers speaks in his paper of excluding cases of inflammatory rheumatism of ordinary severity. In order to comprehend the New York Life results it is necessary to know what is meant by severe attacks of rheumatism. The high mortality experience by the New York Life is evidently due to heart disease and possibly acute infections but it is common knowledge that heart complications occur as frequently in cases of slight or moderate attacks of rheumatism as in the more severe attacks. Dr. Rogers states that the results should not be compared with the experience of carefully selected standard risks but the question naturally arises—"Is it fair to rate cases of severe inflammatory rheumatism where there is no history of involvement of the heart and let those of slight or moderate severity, where there is a history of distinct possibility of some cardiac involvement, go without a rating?" Should not the severe cases be separated into two groups? (1) Those where there is a possibility of involvement of the heart or where there is the presence still existing of a focus of infection; (2) those where there is no possibility of heart involvement and no evidence of focal infection or in which the focus of infection has been eliminated. In the latter group would fall many cases of severe rheumatism but I venture to say that the mortality in this latter group would not be much, if any, higher, than that experienced by the Mutual Life.

Dr. Symonds—Some three or four years ago when we had our joint meeting with the Actuaries, Dr. Rogers and Mr. Hunter presented their scheme of Numerical Ratings, and at that time I took the ground that it was a difficult one to apply because you had to make different allowances for the Medical Examiner—a Medical Examiner who was very skilled would give worse results in heart murmurs than the average Medical

Examiner, because he would pick out only those which were unquestionably heart lesions, and I am inclined to think that the recent experience of the New York Life in Mitral Regurgitation is an exemplification of that fact. I will try and show this on the blackboard.

Let us go back to their 1919 report on Mitral Regurgitation and I do that because they give there more particularly the percentage of the cause of death. If we take normal risks, 100,000 at age 34, the ultimate death rate by the Medico-Actuarial table is five per thousand, and that give 500 deaths, of which according to our Medico-Actuarial reckoning regarding the causes of death, about 8% would be due to heart disease, making forty out of that total.

In 1919 they published some results of Mitral Regurgitation without hypertrophy, giving a mortality of 181%. Translate that again into the same expression of 100,000 risks and we get 905 deaths. These gave a ratio of 37% of deaths from heart disease. This showed that 334.85 of them would be due to heart disease.

In the next group, Mitral Regurgitation with Rheumatism, they showed a mortality of 328%. If we translate this again into our hundred thousand, we get 1,640 deaths and 70% of them were due to heart disease, bringing the total up to 1,148. Now those are the actual figures. Let us assume that we had 100,000 cases all of genuine Mitral Regurgitation, in which the mortality would be 50%. That would give us 2,500 deaths, and when we assume that 75% of them instead of 70% would die of heart disease, it would give 1,875 deaths from that trouble.

Now let us take the mixtures of normal cases and of mitral regurgitation. When the war broke out we had begun to learn a good deal about mitral regurgitation, but with the war we learned an enormous lot more. In fact, Mackenzie of London was so enthusiastic that it almost looked as if a man who had mitral regurgitation was a better risk than the man who did not have it, because he would take care of himself. At any rate we found there were many mitral regurgitant

murmurs which signify practically nothing; there were the cardio-respiratory and a large number of others in which the significance of the so-called mitral regurgitant murmur was of very little consequence. If we take the average run of Examiners who are contributing to our results, some of them will report those murmurs as mitral regurgitant which have very little bearing upon the value of the risk, and these will give a dilution of normal risks with normal hearts in the mass which will materially affect the result. You see what we have here. Here are the normal, and here are the unquestioned mitral regurgitations. If we take 80,000 normal risks they will give us 400 deaths, or five per thousand, 32 of which will be due to heart disease. If we take 20,000 of our genuine Mitral Regurgitations, they will give us 500 deaths with 375 of them due to heart disease, and the total will be 100,000 entrants with 900 actual deaths and 407 deaths due to heart disease. It compares quite closely with our group there—number of deaths 900 as against 905; number of deaths from heart disease a little bit higher—407 against 334.85. If we take 40,000 normal risks they will give us 200 deaths, of which 16 will be due to heart disease. Add to that 60,000 genuine Mitral Regurgitations and they will give us 1,500 deaths, of which 1,125 will be due to heart disease. Adding these up, we get 100,000 entrants with 1,700 actual deaths, a mortality of 340%, and 1,141 of them will be due to heart disease. That group will compare very favorably with our group of Mitral Regurgitations with Rheumatism, and that I think is largely the explanation of the differences.

The following table shows these facts more concisely. All the entrants are supposed to be at the attained age of 34, which gives a normal qx of 5. The first line gives the facts pertaining to 100,000 normal risks. The 2nd, 3d and 4th lines give the facts obtained from the author's paper of 1919. The fifth line gives the facts assumed to be furnished by 100,000 cases of genuine mitral regurgitation due to actual pathological lesions of the mitral valve. The three items following show the results of mixing together certain proportions of normal

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risks and the genuine hypothetical mitral regurgitations. The similarity between these three and the authentic results set forth in the 2nd, 3d and 4th lines is interesting and perhaps instructive.

Entrants and Character	Number of Deaths	Mortality as compared with normal	Deaths due to heart disease	Percentage of these compared to normal
100,000 normal.....	500	100%	40	8%
100,000 M. R. without Hypertrophy.....	905	181%	335	37%
100,000 M. R. with Hypertrophy.....	1,125	225%	450	40%
100,000 M. R. with Rheumatism.....	1,640	328%	1,148	70%
100,000 M. R. genuine (hypothetical).....	2,500	500%	1,875	75%
{80,000 normal.....	{400		{ 32	
{20,000 genuine M. R..	{500		{375	
100,000.....	900	180%	407	45%
{70,000 normal.....	{350		{ 28	
{30,000 genuine M. R..	{750		{562.5	
100,000.....	1,100	220%	590.5	53%
{40,000 normal.....	{ 200		{ 16	
{60,000 genuine M. R..	{1,500		{1,125	
100,000.....	1,700	340%	1,141	67%

In the last group, we are getting a much larger proportion of genuine Mitral Regurgitations, and while I do not want to be too much of a prophet, yet perhaps I can predict that in five years the New York Life will find that their mitral regurgitations without hypertrophy are going to show up bad in their recent business, because the American examiner during the war was well trained in that particular line. He has learnt much, and all of you who are doing sub-standard business will need to bear that in mind, that you are getting better and more accurate reports in heart murmurs now than you ever got before.

Dr. Eakins—When Dr. McMahon honored me by suggesting that I discuss these admirable papers of Mr. Hunter and Dr. Rogers, and Dr. Rockwell, I feared I had already said too much before you on the subject of sub-standard business. We can never hear more than is good for us from sources of authority, or of original investigation. We can, however, become surfeited with discussions which simply agree with our superiors. Finding myself in the position of an agreeing discussor, and having nothing to say concerning hearts or hemoptysis, I denied myself the honor which Dr. McMahon offered, having no wish to satiate you, with nothing. Upon reflection I assumed the privilege of repudiating my renunciation as there is something worthy of being said, which, up to the present, has not been sufficiently emphasized.

The danger of sub-standard business!

Within recent years many Companies have entered the so-called sub-standard field. The fear appears well grounded that some of them are ill-advised in so doing. They seem to consider that under-average risks constitute a very valuable by-product of the business which, regardless, they should have on their books. They seem to forget the fundamental fact that no by-product is commercially worth while unless it can be obtained, and marketed, at a profit. The Essayists will confirm the statement that sub-standard business is not only dangerous but actually constitutes a menace to any Company which does not exercise the utmost caution in its selection. It should not be handled blindly, carelessly, indiscreetly nor indiscriminately. It has a way of exploding once in a while, which is disconcerting when unlooked for or unexpected. This exploding tendency is brought out forcibly by the experience in mitral regurgitation with a history of acute articular rheumatism. Other potential explosions may be found in all lung, heart and kidney cases with low systolic and moderately high diastolic blood pressures.

Never having been told, nor seen any published figures, I do not know what percentage mortality other Companies, issuing insurance on impaired risks, have sustained in that

class alone. Frequently I have been asked, privately, for the experience of my own Company. At the end of 1921 the Reliance general mortality was, on the American Experience Table, 49.8. At the same period its sub-standard mortality was 73.5, for that year. In connection with these percentages please bear in mind that the Company which I represent meticulously scrutinizes, rigidly selects, and timidly issues on, every acceptable sub-standard risk presented to it. It does not permit itself to be drawn into meeting competitive extra ratings for the purpose of beating the other fellow. With a sub-standard mortality of twenty-four percent higher than the Company's general mortality I tremble to think what it would be, were that class of business not selected with the utmost care. It behooves one, attempting to shoulder a load of touchy explosives, to be ever prudent, never forgetting "the danger of sub-standard business."

Dr. Chapin—Mr. President and Gentlemen: As I told Dr. McMahon when he asked me to discuss these papers, the Company which I represent does not sub-standard heart business, so that I have no data to contribute. I am much interested in the subject, however, and have enjoyed as usual the latest study of Dr. Rogers and Mr. Hunter.

The first table given in their study of cases with a history of inflammatory rheumatism deals with the number of attacks and the period within which they occurred. The results given by this table are inconsistent and inconclusive, as the writers point out. I do not think this is surprising, for there is another factor to be considered. It is important, of course, to know how many attacks of rheumatic fever the applicant has had and how recent they have been. It is also important to know whether a focus of infection has been found, and if so whether it has been removed. Both these factors are taken into consideration nowadays in sizing up a risk, and the writers of this paper state the case epigrammatically when they say that in their ratings their standards are more favorable than their experience.

Of more than passing interest is the table showing the im-

portance, or rather unimportance, of ages at entry. The ratings evidently should be the same regardless of age. The high death rate from heart disease among these cases is not surprising, especially as the group studied is that of attacks of inflammatory rheumatism sufficiently severe to require a rating.

The table showing the mortality of applicants with mitral regurgitation and history of rheumatic fever confirms the conclusions drawn for us three years ago by Dr. Rogers and Mr. Hunter. Of particular value is their warning that the hazard involved is much greater than the sum of the separate ratings.

Dr. Rockwell's figures parallel very closely those of the New York Life. In his Group III which is very similar to the group considered in Dr. Rogers' fourth table, the mortality is 429%, as compared with 396% in Dr. Rogers' cases. In Dr. Rockwell's group of 560 cases with history of rheumatism as the only impairment, the mortality is 134%, which is approximately the same as Dr. Rogers' experience with his combined groups with history of rheumatism. The standard ratings of the two companies for mitral regurgitation without history of rheumatism are identical, as are also their ratings for history of rheumatism without heart lesion.

Dr. Rogers—I have listened with much interest to what Dr. Symonds has had to say. His argument is ingenious and, as is usual with him, it was presented clearly and skilfully. There may be something in his suggestion that a certain proportion of our so-called mitral regurgitations may not have been mitral regurgitations at all but I think that the internal evidence is to the effect that there is nothing like the proportion suggested in his argument. Most of you will agree, I believe, that there is a tendency among medical examiners to make no mention of hypertrophy of the heart unless that hypertrophy is clearly marked. Medical examiners either do not find the very slight hypertrophies or, if they find them, make no mention of them. Is it not, therefore, fair to assume that a group of cases of mitral regurgitations with hyper-

trophy will be made up in very large part of cases which belong in that group and with, at the most, a very small admixture of cases that have no hypertrophy? Now these cases of mitral regurgitation with hypertrophy did not show a mortality anything like as high as the 400% or 500% which Dr. Symonds' argument requires. On the contrary, they showed a mortality of 225% or thereabouts. Moreover, this experience corresponds very closely with that of the Equitable, as presented in Dr. Rockwell's excellent paper. Working along very much the same lines, using nearly identical standards, his Company obtained substantially the same results as my own. The difference between us is but slight. In the light of this experience, I am unwilling to abandon our position because of any philosophical considerations, however ingenious, and I shall continue to advise my Company to accept mitral regurgitations along very much the lines indicated by our figures.

I agree with Dr. Symonds that at the present time we are receiving very much better reports on heart lesions than we did formerly, and that the interest of the medical profession in the study of heart murmurs, stimulated as it has been by the World War, will result in much more careful examinations in the future than any we have known up to this time. On the other hand I still hesitate to accept unqualifiedly the dictum of experts in heart lesions for the reason that I have seen them fail often enough to leave in my mind grave doubt of their infallibility.

Dr. Eakins' contribution to the discussion is of real value. His Company's experience with sub-standard business is much more favorable, I suspect, than will be the experience of many Companies that have recently undertaken work in that field. Indeed, any Life Company should undertake a sub-standard business in a spirit of real conservatism. It is quite true that the New York Life has been engaged in a very large sub-standard business for more than 25 years and that we have been securing results right along during that period well within 10% of our predictions—Mr. Hunter will correct me if I am

not accurate in this statement—but it must be remembered that we have kept our sub-standard business constantly under observation and whenever, in any group, we found ourselves slipping below the safety line, we have shifted our position so as to correct that error.

The figures submitted by the Mutual Life show that, selected with considerable care, it is possible to obtain a substantially normal mortality among cases of acute articular rheumatism. I am confident that cases of the same sort which we have insured in my own Company on standard plans would yield a similar result, but if, after a Life Company has placed in its standard class the carefully selected risks, it wishes to take the risks that are excluded from that class, it must provide for a much higher mortality among them. Indeed, I believe that among these less desirable risks it will experience a mortality somewhat along the lines indicated in our paper. I think that is true with regard to any other impairment. I remember that one of my colleagues on one occasion announced that he was getting a mortality among syphilitics of somewhere in the neighborhood of 100%. It may very well be that, by exercising extraordinary care, a small group of such risks may be gotten together which will produce a low mortality but, taking them by and large, from all parts of the country, I am sure that no Company will succeed in securing a normal mortality among them. It is true that the experiences of our respective offices may differ somewhat in almost every group we analyze, but I cannot think these differences will be very substantial in most of the types we are interested in. This discussion brings out clearly the fact that with regard to the value of these statistics, the several conclusions that we draw differ very considerably. For my own part, I find considerable support for the method of numerical ratings. Dr. Rockwell's Company, using substantially the same tools, produces about the same results. Indeed, I do not believe that there is a single Company represented here to-day which, having used the numerical method of selection, would ever abandon it for any other that has so far been suggested. It

steadies the individual judgment and enables us to make a fine distinction between those risks which should be placed in the standard class, and those that must be accepted as under-average lives. Personally, I am satisfied that the numerical values which have been brought out here this afternoon will serve to steady the judgment of each one of us when it comes to dealing with rheumatisms hereafter. That is the very essence of what the numerical method means. It does not mean that every selector shall slavishly follow numbers. It rather means that he shall use numbers to steady his judgment and to eliminate that personal equation which has always been so mischievous in medical selection in life insurance.

Mr. Hunter—When the figures regarding Mitral Regurgitation with rheumatism were presented to our medical Board, I was met with the statement that it could not be so, that we could not have as high a mortality as was indicated—the mortality being in the neighborhood of 300%; but the Equitable showed a mortality somewhere in the neighborhood of 400%, which is a pretty good proof of the dependability of our figures.

Most of the doctors have their own point of view with regard to the deductions which might be made from our experience, and I hesitate as a layman to give my opinion. It seems to me that if a high mortality is experienced among the Mitral Regurgitations with articular rheumatism, then the persons who have rheumatism have a serious type of heart murmur, and that functional murmurs are not included by mistake. Some of our medical men seem to think that a large proportion of the Mitral Regurgitations were due to rheumatism. We accordingly investigated 800 cases—400 with hypertrophy and 400 without hypertrophy—and we found that 4% only of the cases showed a history of rheumatism of such a nature that they were rated for that history. In addition there were 10% of the cases in which the attack was so remote that no rating had been put on it, so that there were 14% of all Mitral Regurgitations which showed a history of rheumatism at any time from childhood on. Of course, I realize that there may

be a large number of cases of rheumatism which were not mentioned in the application, but, so far as our records go, only 14% showed a history of rheumatism.

So far as the mortality from Mitral Regurgitation is concerned, I entirely agree with Dr. Rogers. We have had such a wide experience—which has been substantiated by the Equitable—that our figures may be accepted as reasonably accurate. Please bear this in mind, that while we may have a slightly higher mortality in the future on account of the more careful selection the mortality under the Mitral Regurgitations decreases with advancing policy years. If the relative mortality were 200% for the last five years it might run down in the course of fifteen years to 150%. The early supposition of the Equitable that there would be a delayed mortality on Mitral Regurgitation is not borne out by experience—it is just the reverse. I should like also to say that the conditions which were found in connection with Mitral Regurgitation follow in the majority of other types of impaired risks, *i. e.*, the relative mortality generally decreases with the advancing policy years.

Dr. Rogers is very modest in claiming that we come within 10% of our predictions on our sub-standard business. We have just made up our mortality experience for all cases issued with an advance in age since 1906. In measuring this experience we assume that the risk is of the same quality as a standard risk at the advanced age—that is to say, if a man is 30, and rated up to age 40 in determining the expected mortality we would take the rate of mortality at age 40. In the early years of this business we had a relative mortality of 10% higher than in the standard business; but at the end of 1921 we were within 1% of our predictions. This is partly accidental and we do not expect that our experience will always be so close to the expected.

Dr. McMahon—Dr. Rockwell will close the discussion of his paper.

Dr. Rockwell—Mr. President: I have little to say except perhaps to tell you how these figures of the Equitable were

compiled. We were engaged in casting a general mortality of the experience of the Company. After that had been concluded the data of Messrs. Rogers and Hunter paper came to my attention. Noting that some work had already been done by us on special classes, I asked our statistician if he would cast the Equitable's experience with mitral regurgitant murmurs, with or without hypertrophy, and with or without a history of rheumatism. He did so, and on comparison we learned that while the figures were of course not identical, they pointed very markedly in the same direction, particularly as to the degree of hypertrophy. Our present method of rating the degree of hypertrophy extends only over a period of two or three years. We all remember the time when an examiner would report that the applicant has a slight mitral regurgitant murmur, "well compensated" by which he would mean an appropriate degree of hypertrophy and that the heart was taking care of itself very well; so we thought those were very good cases to take. We started in 1900 to rate those cases on an average of plus 40 to plus 50, expecting a deferred mortality, but we found we were having an occasional early claim. Six or eight years afterwards, we made a study and decided to more than double our ratio of rating, which at the present time is from plus 75 to plus 150, if at all acceptable.

Now the question comes up what to do with applicants who present a history of rheumatism and heart murmur. The Canadian gentlemen have concluded that it is a pretty bad combination and are declining applicants who present a history of rheumatism along with the murmur.

So far as rheumatism itself is concerned, our class of substandard cases was rather small. I did not present the cases of rheumatism taken in the standard class, because I am bound to confess they did very well, but simply presented those taken substandard. Undoubtedly those were cases of well marked sharp attacks of severe inflammatory rheumatism. The mortality of 134 in that class also falls in line with the New York Life's experience.

Knight—Overweight and Underweight 193

Dr. Knight, of the Metropolitan Life Insurance Company, distributed and described a set of new tables, giving the limits of overweight and underweight corresponding to various mortality ratios. These tables are now being used by the medical and lay reviewers in the Home Office of the Metropolitan.

TABLES OF OVERWEIGHT AND UNDERWEIGHT CORRESPONDING TO VARIOUS MORTALITY RATIOS

BY A. S. KNIGHT, M. D.

Medical Director, Metropolitan Life Insurance Company

We have recently constructed two tables giving the limits of overweight and of underweight corresponding to various mortality ratios which, we think, will be very convenient for use of the medical and lay reviewers of applications in our Home Office.* As you will see, we have chosen the mortality ratios 100, 110, 120, 125, 135, 140, 150 and 160, each of them on the M. A. table. These values correspond to the mortality limits we put on various plans of insurance. Thus, we do not usually issue ordinary policies on those whose mortality may be expected to go beyond 125 of the M. A. table; nor intermediate insurance on those whose expected mortality is above 150 per cent., etc. For each one of these ratios, the table shows a limiting weight for each age period and for each inch of height. Any one should then be able to see at a glance in what mortality class an applicant falls according to his weight, having in mind his age and height.

The tables in the more recent report of the Medico-Actuarial Committee, 1918, entitled, "Standard Mortality Ratios Incident to Variations in Height and Weight Among Men," it will be remembered, were limited to three height groups, namely, short men, men of medium height and tall men. This is a distinct disadvantage, for reviewers need the figures for the actual inch of height. Our overweight table has attempted

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to remedy this defect and gives values for each inch of height from 5' 1" to 6' 2". It must be clear, however, that our table has been constructed from the previous M. A. material. No attempt has been made at correction of the original data, our purpose being entirely to give the more useful values for each inch of height. We make no pretense at greater accuracy than the original source.

The method used in compiling the table was essentially as follows:

We considered each mortality ratio and each age group separately. Thus, we took the conditions in the mortality ratio 120 for each of the age groups 20 to 24, 25 to 29, 30 to 34, and so on. For each age group, a graph was constructed showing the number of pounds overweight at each inch of height from 5' 1" to 6' 2". The construction of this graph was made difficult by the fact that we knew the position of only three points on it, namely, at 5' 5", 5' 8.5", and 5' 11.8". These are the average heights of each one of the three height classes as reported by the Medico-Actuarial Committee in 1912. After considerable preliminary work, comparing the relations of the three points in the several age classes, it became clear to us that the best curve through the several points would be a common parabola. Two intermediate points on the curve were decided upon which were most consistent with known facts and which gave a parabola whose extremes gave reasonable values of overweight. The number of pounds overweight was then read off for each inch of height on the curve. These overweight values were added to the average weights appearing in the Medico-Actuarial build-table and the results are presented herewith.

When the table was completed for each mortality ratio, care was taken to make them mutually consistent and to eliminate the few irregularities which arose in the course of the work. These changes never involved the addition or subtraction of more than a pound or two.

We believe that we have constructed a table which is essentially identical with that of the M. A. Report, 1918, but

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which has added advantage of giving the values for each inch of height and of greater convenience for use because it gives, at a glance, the limiting weight corresponding to any mortality ratio rather than the amount of overweight which has to be added to the average weight to give the significant figure. The few liberties we have taken with the original material are more than compensated for by the convenience of the results.

Our table on comparison with the New York Life build-table appears to be more severe to overweights. The differences are most marked at the younger ages, for the extremes of overweight and especially for tall men. Some of this difference is due to the fact that the New York Life experience is based on the expected mortality values of the Compound Progressive Table. The latter table has considerably higher death rates than the more recent M. A. table. There is also good evidence that the New York Life ratings for the tall men are too liberal. In any case, we have felt justified in using a somewhat severer measure because our business is written virtually on a non-participating basis, which leaves little leeway of safety in adjusting the experience on sub-standard lives. We recognize that there is still very much left to do in connection with the mortality of overweights and we shall carefully follow our experience on this new basis and will report to you from time to time how closely the new table corresponds to the fact of our actual experience.

The underweight table is very clearly a much less important matter. Underweight is hardly worthy of consideration in insurance selection after age 35; and, even at the younger ages, this impairment is largely limited in significance to the taller men. Underweight rarely affects the type of ordinary insurance which is granted to shorter men. Among tall men, underweight may result in a mortality of 125 to 135 per cent. according to the M. A. table but then only at the ages under 30. Except in extreme cases which are rarely met with, underweight when uncomplicated by any other impairment is very obviously an asset rather than a detriment and needs very little attention in insurance medical selection.

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LIMITS OF OVERWEIGHT CORRESPONDING TO VARIOUS MORTALITY RATIOS*

Heights	Mortality Ratios	Weights According to Age Period									
		20-24	25-29	30-34	35-39	40-44	45-49	50-53	54-56	57-59	
5 ft., 1 in.	100	144	137	131	129	128	130	131	130	129	
	110	160	149	138	138	140	140	142	142	142	
	120	166	159	147	149	150	151	151	152	152	
	125	167	160	154	152	152	154	156	157	157	
	135	173	166	161	159	159	161	162	163	163	
	140	177	170	165	162	163	163	166	166	168	
	150	182	175	167	168	169	170	171	174	174	
	160	187	183	178	175	175	176	177	181	181	
5 ft., 2 in.	100	149	141	135	134	133	135	135	135	134	
	110	163	152	144	143	145	145	147	147	147	
	120	169	162	151	153	155	155	155	156	156	
	125	171	163	158	157	156	158	160	161	161	
	135	177	170	165	163	163	165	167	167	167	
	140	180	173	168	166	166	168	170	171	172	
	150	187	179	172	172	173	175	176	178	178	
	160	192	187	181	179	178	180	181	185	185	
5 ft., 3 in.	100	153	146	141	140	139	141	141	141	140	
	110	166	157	150	149	150	151	153	153	153	
	120	173	166	157	158	160	160	161	162	162	
	125	175	168	163	160	161	163	165	165	166	
	135	181	175	170	168	168	170	172	173	174	
	140	185	178	173	171	171	173	175	176	177	
	150	191	183	177	177	178	180	181	183	183	
	160	197	191	186	184	183	186	187	190	191	
5 ft., 4 in.	100	158	151	147	147	146	147	147	147	146	
	110	169	161	156	156	155	157	158	159	159	
	120	177	170	163	164	165	165	167	168	168	
	125	180	173	168	167	167	169	171	171	172	
	135	186	180	175	174	173	176	178	179	180	
	140	190	183	178	177	176	179	181	182	183	
	150	196	188	183	183	183	185	187	189	189	
	160	202	196	191	190	188	191	193	196	197	
5 ft., 5 in.	100	163	156	153	153	152	154	154	154	152	
	110	173	166	162	162	162	163	164	165	165	
	120	182	174	170	170	171	171	173	174	174	
	125	185	178	174	173	173	175	177	178	178	
	135	191	185	181	180	179	182	184	185	186	
	140	195	188	184	183	182	185	187	188	189	
	150	201	194	190	190	190	191	194	196	196	
	160	207	201	197	196	194	197	200	202	204	

* Based on Medico-Actuarial Reports, 1912-1918.

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Heights	Mortality Ratios	Weights According to Age Period									
		20-24	25-29	30-34	35-39	40-44	45-49	50-53	54-56	57-59	
5 ft., 6 in.	100	166	160	159	159	159	160	161	161	161	
	110	177	170	168	168	168	170	171	172	173	
	120	186	179	176	176	176	178	180	181	182	
	125	189	183	180	179	179	181	183	185	185	
	135	196	190	187	186	185	188	190	192	193	
	140	200	193	190	189	188	191	193	195	196	
	150	206	200	197	196	196	197	200	202	203	
	160	212	206	203	202	200	203	206	209	210	
5 ft., 7 in.	100	168	164	164	164	165	166	167	168	168	
	110	179	175	173	173	174	176	177	178	179	
	120	189	183	181	181	182	184	186	187	188	
	125	192	188	186	185	185	187	189	191	192	
	135	199	195	193	192	191	194	196	198	199	
	140	203	198	196	195	194	197	199	201	202	
	150	209	205	203	201	201	203	206	208	209	
	160	215	211	208	207	206	209	212	215	216	
5 ft., 8 in.	100	170	167	168	169	170	172	173	175	175	
	110	181	178	178	179	179	182	183	184	185	
	120	191	187	186	187	188	190	192	193	194	
	125	195	192	191	190	191	193	196	197	198	
	135	203	199	198	198	198	200	203	204	205	
	140	207	202	201	201	201	203	206	207	208	
	150	212	209	208	207	207	209	212	214	215	
	160	218	215	213	213	212	215	218	221	223	
5 ft., 9 in.	100	173	171	171	173	174	176	177	179	180	
	110	182	180	181	184	184	186	188	189	191	
	120	192	189	189	192	192	194	196	197	199	
	125	196	194	193	194	195	198	200	202	203	
	135	205	202	202	202	203	205	208	209	210	
	140	209	206	205	205	206	208	211	212	214	
	150	214	212	211	211	211	215	217	219	221	
	160	221	218	217	217	217	220	223	226	228	
5 ft., 10 in.	100	176	175	175	176	177	180	181	183	184	
	110	181	181	184	187	188	190	192	193	195	
	120	192	191	192	195	196	197	199	201	203	
	125	197	195	196	198	199	202	204	206	208	
	135	206	204	204	206	207	209	213	214	215	
	140	210	208	208	209	210	212	216	217	219	
	150	216	215	214	214	215	219	222	224	226	
	160	223	221	221	221	221	225	228	231	233	

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Heights	Mortality Ratios	Weights According to Age Period									
		20-24	25-29	30-34	35-39	40-44	45-49	50-53	54-56	57-59	
5 ft., 11 in.	100	†	†	†	†	179	183	185	187	189	
	110	180	182	186	189	192	194	196	197	199	
	120	193	193	194	198	199	201	204	206	208	
	125	198	197	199	201	203	206	208	211	213	
	135	207	205	206	209	210	213	216	219	220	
	140	211	209	210	212	213	217	220	222	224	
	150	218	217	216	217	219	223	227	229	232	
	160	225	224	224	224	225	230	233	236	238	
6 ft., 0 in.	100	†	†	†	†	181	186	189	190	192	
	110	179	183	188	191	195	197	200	201	203	
	120	193	195	196	201	203	205	208	211	213	
	125	199	199	201	204	207	210	213	216	218	
	135	208	207	208	212	214	217	219	223	225	
	140	213	211	212	215	216	221	223	226	229	
	150	220	219	219	221	223	227	231	233	236	
	160	227	227	227	228	229	234	238	241	243	
6 ft., 1 in.	100	†	†	†	†	184	190	193	194	196	
	110	180	185	191	194	198	202	204	205	208	
	120	195	199	200	204	207	210	213	217	219	
	125	201	201	204	207	211	215	218	222	224	
	135	209	208	209	212	217	222	223	228	231	
	140	216	213	215	218	221	226	228	232	235	
	150	223	222	222	224	226	231	235	238	241	
	160	229	230	230	231	232	238	243	246	249	
6 ft., 2 in. and over	100	†	†	†	†	188	193	199	200	202	
	110	180	187	193	197	201	205	208	210	212	
	120	196	200	202	209	213	215	219	222	225	
	125	202	204	207	211	216	219	223	227	229	
	135	210	211	212	216	221	227	228	232	236	
	140	218	216	217	221	225	230	232	235	240	
	150	225	225	225	228	230	236	239	244	246	
	160	231	233	233	236	237	244	248	252	254	

† At these heights and ages, all weights are over 100% mortality.

DIRECTIONS

Use for males and for females.
Drop fractions of an inch, $\frac{1}{2}$ -inch or less; over $\frac{1}{2}$ -inch, use next inch.
Overweights whose abdominal girth exceeds the expanded chest girth should receive additions to mortality ratios as per table on bottom of next page.

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LIMITS OF UNDERWEIGHT CORRESPONDING TO VARIOUS MORTALITY RATIOS¹

Heights	Mortality Ratios	Weights According to Age Periods			
		20-24	25-29	30-34	35-39
5 ft., 3 in. to 5 ft., 6 in. incl.	100 110	112 99	110 *	108 *	* *
5 ft., 7 in. to 5 ft., 10 in. incl.	100 110 120 125	139 125 114 110	139 121 110 *	139 116 * *	127 * * *
5 ft., 11 in. to 6 ft., 2 in. incl.	100 110 120 125 135	† 158 143 137 126	† 152 136 130 *	† 141 * * *	† * * * *

ADDITIONS TO MORTALITY RATIOS FOR OVERWEIGHTS WITH EXCESS ABDOMINAL GIRTHS

Abdominal Girth	Ratios, 100% to 140% incl.			Ratios, 150% and 160%		
	Under Age 40	Age 40-50	Age 50 and over	Under Age 40	Age 40-50	Age 50 and over
1-inch excess.....	0	0	5	0	5	10
2-inch excess.....	0	5	10	5	10	15
3-inch excess.....	5	10	15	10	20	25
4-inch excess.....	10	15	25	20	30	40

¹ Based on Medico-Actuarial Reports, 1912-1918.

* Where no values are given in the above table, there is no restriction on underweights who are good risks in other respects. There is also no restriction on underweights over age 40.

† At no weight under age 40 does the mortality on tall men go as low as 100.

SECOND DAY

President McMahon in chair. The meeting was called to order promptly at ten o'clock A.M.

The Secretary announced that he had cast a ballot as instructed for the officers and members of the Executive Council whose names were placed in nomination the day preceding. These officers and members of the Executive Council were declared elected as follows:

PRESIDENT

DR. FRANK L. GROSVENOR

FIRST VICE-PRESIDENT

DR. WILLIAM R. WARD

SECOND VICE-PRESIDENT

DR. CHESTER F. S. WHITNEY

SECRETARY

DR. ANGIER B. HOBBS

TREASURER

DR. CHARLES L. CHRISTIERNIN

Election of Officers

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EDITOR OF THE PROCEEDINGS

DR. ROBERT M. DALEY

MEMBERS OF THE EXECUTIVE COUNCIL

DR. G. A. VAN WAGENEN

DR. EDWIN W. DWIGHT

DR. J. ALLEN PATTON

DR. W. W. BECKETT

DR. F. L. B. JENNEY

The Secretary read a letter from Dr. Daley in which he called attention to the need of a revision of some of the articles in the Constitution and By-Laws. The Secretary moved that a Committee of Three be appointed to review the Constitution and By-Laws and report at the next meeting any changes or modifications that may seem necessary or desirable. Dr. Rockwell seconded the motion and it was carried.

The President appointed Dr. Daley, Dr. Hobbs and Dr. Porter, a Committee of Three to review the Constitution and By-Laws, with Dr. Daley as Chairman.

Dr. Fisher—I have here some data on rheumatism which may be of interest to the Association.

In Dr. Rogers' and Mr. Hunter's paper, they give a record of 5,876 cases with a history of inflammatory rheumatism which show a mortality of from 45 to 60 points in excess of the M. A. Table, and from 35 to 50 points in excess of the experience on standard risks.

Dr. Rockwell, in his paper, furnishes a record of 560 sub-standard policies, with a history of rheumatism as the only impairment, with 29 actual claims; one of these a war claim

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and two, epidemic claims. Both companies received an extra premium, as indicated.

The Northwestern Mutual issued 7,831 policies during the years 1893-1915, both inclusive, with a previous history of rheumatism.

Six thousand fifty-one of these were issues of 1893-1907, inclusive, our mortality upon this group being 7 points better than the general mortality experience of the company by the Actuaries' Table. We expected 402 deaths, as provided for by the table. Actual deaths, 199.

The material was divided according to the number of attacks and occurrence of attack prior to examination.

NORTHWESTERN MORTALITY EXPERIENCE—ACTUARIES' TABLE

HISTORY OF RHEUMATISM—ONE ATTACK

	<i>Lives</i>	<i>Deaths</i>		<i>Points More (+) or Less (-) than General Average Mortality</i>
		<i>Expected</i>	<i>Actual</i>	
Two years and less prior...	503	28	14	- 6
Between 2 and 5 years prior	888	50	14	-28
Over 5 years prior.....	4150	283	148	- 4
Total.....	5541	361	176	- 7

HISTORY OF RHEUMATISM—TWO OR MORE ATTACKS

	510	41	23	<i>General Average Mortality</i>
Total.....	6051	401	199	- 7

We computed our mortality with respect to history of rheumatism, by the A. M. Table, covering issues of 1906-1915, carried to the anniversary in 1920, upon 1,780 policies. Actual deaths, 68. Ratio %, 64.27.

Introduction—Intra-Thoracic Pressure 203

Dr. McMahon—The first subject for discussion this morning is one which I am sure will prove most interesting to all of the members. Dr. Frost, of the New England Mutual Life Insurance Company, will give us a demonstration of a suggestive method for testing cardiac resiliency. Before Dr. Frost presents his paper, however, Dr. Dwight will make some introductory remarks.

Dr. Dwight—In introducing this subject I wish first to say, that this is simply a report of progress. We are reporting a test which we have studied for only a comparatively short time and the value of which is not yet fully determined even in our own minds. We believe it is suggestive and to be worthy of further study.

I am not going to waste the time of the Association in discussing the need of some such test—I think that is clear in the mind of every Medical Director, and of every one to whom these problems of circulatory disease—using the term in its broadest sense—are brought up from time to time.

We are learning a great deal in regard to hearts and circulation. As the result of the war we have had very clearly brought to our attention the importance, for many reasons, of the application of a load to the heart muscle, in demonstrating its condition. The tests introduced by the Surgeon-General are of great value, but as applied to Life Insurance it seems to me that they are of doubtful value, as our problems are very different from those of the Army. In selecting soldiers, one pays very little attention to what is going to happen twenty years hence. He considers, rather, ability to handle a load for a comparatively short period. When measured by Life Insurance standards, further objections are raised by the fact that the Army tests apply only to a group of young men, most of whom are apparently healthy. They are really an attempt to locate the physical or man-power of the country able to endure continued effort for a brief period—from one to five

years. We look at it from a very different point of view. The spread is much wider, and most of us are considering cases with doubtful circulatory conditions extending over a period of from below age 20 to above age 60. We meet men of all types, and all habits of life. It is, I think, sufficiently obvious to all of us, that the effect on the circulation of jumping on one foot fifty times, or one hundred and fifty times, varies not only with the condition of health of the individual, but with his training for this particular effort.

In common with other Companies and other men, we have felt the need of a test of the strength or reserve power of the heart muscle. This is a report of progress along these lines, and the idea which I had in suggesting some such method was, first, that we must have a load which could be applied to the circulation as a whole; that load must be susceptible to definite measurement and charting; and the results of the attempt to carry that load must also be measurable and subject to charting. A good many difficulties appeared at the outset, and it was only last year that an idea struck me. Dr. Frost was chosen by me to do this work at our office, for three reasons: First, that he had no preconceived ideas of circulatory conditions from the insurance point of view; second, that I had great confidence in his judicial attitude of mind towards problems which came up, and third, he had the interest and ability to follow through. I wanted to eliminate my own personal equation. I might, or might not, be prejudiced in favor of my own convictions, and I wanted a man who was big enough to tell me I was wrong and that there was nothing in it, if he felt this to be so.

Neither Dr. Frost nor I have the remotest idea that we have developed, in the course of a few months, an entirely satisfactory, accurate and easily applied method by which we can reach a definite or final decision as to the real condition of the myocardium in all doubtful cases. I have followed in this work the same general lines which we followed in connection with the urine some twenty years ago and which brought me into considerable discord with this Association—with our

Introduction—Intra-Thoracic Pressure 205

agents and with everybody else. The criticism was that it was impossible for us to devise in our work an absolutely scientific test by which we could say that the albumin found in a given man's urine did or did not mean anything. We were getting a great many albumins and casts and we knew that a great many of those individuals were not diseased, that their mortality was not excessive. We also knew that if we took any very large number of such cases at ordinary rates, we should get into trouble, so we tried to apply a load to the kidney, believing that, if the kidney could pass a perfectly normal urine when it was submitted to that load, it would be a sufficiently normal kidney for our purposes. We tried it tentatively, we tried it carefully, and we have tried it persistently for nearly twenty years. I am entirely satisfied that the load which we apply to the kidney is sufficient for our purposes. I am making no claim that it is scientific or ultimately perfect, but—the test has worked and is still working.

Now we are trying to do a very similar thing to the heart and circulation. Dr. Frost has worked this out. It is a measurable load which can be applied to the heart and circulation—how much to the heart and how much to other portions of the circulation, I am not prepared to say. It is a measurable load which can be put down in figures, and which will give us a definite idea as to the amount of load which that particular individual is carrying when he develops a certain reaction. The results of carrying that load can also be measured and put down in figures, so that we can say that an individual, carrying this load, accomplished this result.

It has a further advantage, that it can be applied, we believe, with equal facility and with equal comfort to the successful man of affairs applying for large amounts of insurance, who has been working over-hard and not taking time for exercise, or for keeping himself in good physical condition; to the young high-school graduate, the high-school athlete, or to the professional or semi-professional athletes who have been trained along certain lines. It can be applied to any man or any woman, while seated at the examiner's desk, without putting him or her

through any effort which appears either excessive or to make a fool of the individual. I know many men and women who would hesitate about jumping 150 times, or going through setting-up exercises for a strange young doctor. I have never yet found anybody who objected to this process, and it can be accomplished in not over twenty minutes.

It has one manifest disadvantage—besides the fact that we are not yet capable of making what to my mind is surely accurate interpretation—and that is, that I do not believe that it is now or ever will be a safe test to put in the hands of all Medical Examiners or of all doctors. After working at it for about a year, we have only three men at the Home Office who make the test. We had our Chief Examiner from New York City come over and spend a week with us, and he is now using it. That is as far as we have gone. One other point: I cannot say that this is a test which should be applied in hospitals or to individuals who are actually sick or who believe, or have reason to believe, that they have serious trouble with the heart or circulation. That would be an experiment which we are not ready to recommend.

At the conclusion of Dr. Frost's paper, he will be glad to demonstrate just how the test is made and what it shows to any of the gentlemen who wish to try it.

We had a meeting of our general agents in Boston recently, and I asked them to submit themselves to this examination. They responded wonderfully well. I told them I wished first of all to impress upon them that this is no unusual or terrible thing, and also that we are applying it only in doubtful cases, which are now being declined, with the idea of separating out from that group certain individuals who may be accepted for insurance. I also said I wished to put them in such a position that they could go back to their agents, their examiners and their applicants and impress them with the fact that there is nothing in the test to trouble anyone. They did it. I added one thing which I wish to add here. I said, "If there is any man present who has the slightest idea that he has any trouble with his circulation, I do not want to have him take

the test." I remember very well that when the sphygmomanometer was first demonstrated before this Association, we had a considerable number of men of more or less advanced age who had good reason to believe that they did have some trouble, and they wanted to know if it was really so. Unfortunately, we found several with a blood pressure running up to 240-250 and above, and at least two of them went home and died directly, I believe, because of the fact that that test took away all hope. I do not believe this test has any such danger but still I hope you won't try it if you have any doubt about your physical condition.

A STUDY OF CARDIO-VASCULAR REACTION TO
ABNORMAL VARIATIONS OF INTRA-
THORACIC PRESSURE

BY HAROLD M. FROST

*Medical Examiner, New England Mutual Life Insurance Co.,
Boston, Mass.*

It is possible, as a rule, by proper methods of inspection, palpation, percussion and auscultation, aided by instrumental technique, to detect definite cardiac disease; even to define its exact location. The terminal stage of such disease, evidenced by the signs and symptoms of cardiac decompensation, obviously points to muscular insufficiency. In former years, the tendency among clinicians was to emphasize the location and extent of the cardiac lesion. To-day, however, more stress is being laid upon the functional capacity of the heart, upon the reserve power at its command to withstand abnormal strain. As a logical sequence, various methods have been proposed for measuring its functional power.

There is a certain class of cases, particularly within the scope of cardio-vascular degenerations, in which the onset of disease, the incidence of diminishing functional power, may not be detected by the usual methods of examination. There

are many individuals, apparently in good health, who present, without other signs of cardiac disease, an irregularity of rhythm; or a murmur of the so-called "functional" type; or a blood pressure higher or lower than is usually conceded to be normal for their age and environment; or an unusual irritability with a rapid and extensive reaction to muscular exertion or psychic influence. Obviously, it is of the utmost importance to be able to determine whether these conditions indicate an early stage of disease, presage its development or are merely negligible incidents in the working of a normal cardio-vascular system.

From physiological investigation, it has been discovered that the function of cardiac muscle is complex, presenting at least five subdivisions: (1) the power of originating contractile impulses; (2) excitability, the power of receiving impulses; (3) conductivity, the power of conducting impulses by special differentiated fiber paths; (4) contractility; (5) tonicity. Any method of measuring the total cardiac function, to be perfect, should make it possible to estimate each of the subdivisions. Such a method has not yet been presented, although it is possible by instrumental means to detect certain deficiencies of muscle function. The methods so far proposed have been based, in the main, upon the reaction of the heart to muscular exertion, as indicated by changes in pulse rate and blood pressure. These will be briefly reviewed.

In 1894, Christ (1) originated a steppage machine, with provision for measuring the amount of work done, registering the pulse after exertion with the sphygmograph.

The staircase test, proposed by Selig (2) in 1905, involves the rapid ascent of a definite number of steps, with comparison of pulse rate and systolic pressure before and after. The weight of the individual, the height and number of steps being known, it is possible to determine the amount of work done.

Graupner (3) employed the Zuntz ergometer of the bicycle or weight-and-pulley type, with provision for measuring the amount of work done.

Mendelsohn (4) noted the effect on pulse rate of change

from the vertical to horizontal position, completing the test by determining the facility with which the pulse rate returned to normal after a given amount of work.

Other muscular tests comprise the use of dumbbells (5); hopping on one foot a given number of paces (6, 7); contraction of the forearm muscles, at the same time flexing and extending the forearm, antagonizing each movement as much as possible (8); a combination of flexion-extension of the forearm against resistance, abduction-adduction of the thighs and of extended lower limbs (9).

The Katzenstein test (10) involves the compression of the femoral arteries by digital pressure or tourniquet, with notation of the change in pulse rate and blood pressure.

In the Russian test (11, 12) the cardiac function is determined by the length of the period over which the individual is able to hold his breath.

In the foregoing tests, the reaction of a normally functioning heart is indicated by an initial increase of pulse rate with rapid return to normal and a rise in systolic pressure with gradual return to normal; the height of both reactions being in direct proportion to the amount of work done. A delayed increase in pressure, absence of increase or reduction in pressure, with a sustained increase in pulse rate are considered evidence of impaired function.

Another type of test makes use of the cardiac reflexes, in which contraction of the left ventricle is produced by precordial friction, and dilation of the right ventricle by friction over the median line of the abdomen. These changes are determined by percussion or X-Ray (13, 14, 15, 16).

The Koranyi test (17, 18) involves the power of elimination of an excess of sodium chloride, defective elimination indicating cardiac insufficiency.

With the advent of the sphygmomanometer other methods of determining functional capacity were proposed, based on blood pressure readings.

Tiegerstedt (19) advocated a cardiac efficiency factor based upon the velocity-work ratio. Assuming the velocity to

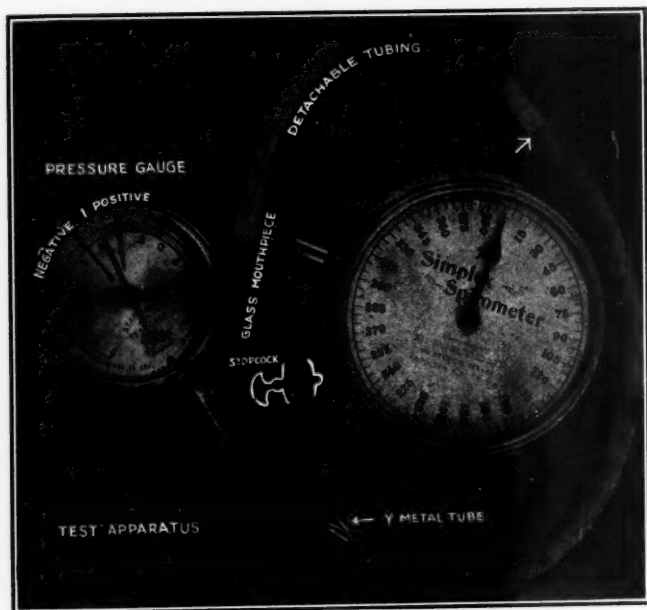
equal the product of pulse pressure and pulse rate, and the work to equal the product of systolic pressure and pulse rate, he found that in the apparently normal heart the ratio of velocity to work is as one to three. Stated more simply, he believed the ratio of pulse pressure to systolic pressure, the blood pressure co-efficient, should be roughly as one to three, normally varying, as expressed in percentages, from 25% to 35%. Any variations from these limits he considered an indication of diminished functional capacity.

Goodman and Howell (20) developed the cardiac strength-cardiac weakness ratio, based upon the duration of the four phases of auscultatory estimation and their relations to each other and to the pulse pressure. Estimating the average duration of phases one to four to be 14 mm. 20 mm. 5 mm. and 6 mm. they believed that cardiac strength, the sum of phases two and three, is to cardiac weakness, the sum of phases one and four, as 55.5 is to 44.4; any increase in the cardiac weakness indicating, of course, a decrease in functional capacity.

Stone (21) believed that the myocardial load, expressed by the ratio of pulse pressure to diastolic pressure, should be normally as one to two, or 50%. Any marked reduction he considered an indication of cardiac weakness. On the other hand, a marked increase he considered a sign of over-load, a possible danger signal.

All of these tests have been checked by various clinical investigators. Some of them, such as the hopping and staircase exercise, change of posture and blood pressure phase relationships, are in common use and of course may be easily applied in the examination of Life Insurance applicants. To the present, however, no test has been developed simple enough to invite universal use and at the same time permitting the examiner to observe the reaction of the cardio-vascular system throughout the entire period in which it is subjected to abnormal strain.

Dr. Edwin W. Dwight, Medical Director of the New England Mutual Life Insurance Company, has long appreciated the need of a more adaptable test of cardio-vascular function.



Test Apparatus

He evolved the idea of determining the effect of changes in intrathoracic pressure upon the heart and blood vessels, as indicated by changes of heart rate and rhythm, and of blood pressure. He had noted that a considerable change in systolic pressure resulted from blowing steadily against resistance. He kindly invited me to assist in the investigation.

Two essentials of technique were at once apparent: (1) that it should be capable of easy application, not encumbered by clumsy apparatus; (2) that it should permit of continuous observation of blood pressure, pulse rate and rhythm.

The apparatus finally decided upon consists of a compound steam gauge and a Simplex spirometer. The gauge is necessarily delicate, registering but five pounds positive or negative pressure. The gauge and spirometer are connected by means of a Y metal tube to a piece of detachable rubber tubing and glass mouthpiece. The latter are detachable for purpose of sterilization by boiling. Between the Y tube and spirometer a stop-cock is inserted, in order that the spirometer may be cut off at will. It is, of course, recognized that this type of spirometer will become inaccurate from continued use in so far as the correct determination of vital capacity is concerned. However, as our object is not to determine the vital capacity but to have the subject expire to his full capacity, this spirometer suffices. The whole apparatus is compact, easily carried about and may be used with the subject sitting at the desk.

Our present technique is as follows. The patient sits at the desk with the apparatus lying before him. A preliminary notation of physical signs, of systolic and diastolic blood pressure and of pulse rate and rhythm are made. The patient inspires as deeply as possible, holding his breath by closing the glottis and allowing the diaphragm and chest to relax against the inflated lungs. The change in systolic pressure is noted until after the intrapulmonary air is released. The variations of pulse rate and tonal rhythm are likewise noted. After an interval of about ten seconds, the subject expires as far as possible and maintains full expiration by closing the glottis and allowing the chest and diaphragm to relax. After

another ten-second interval, the subject blows against the gauge, the spirometer being cut off by the stop-cock. A positive pressure of approximately one pound is maintained for about ten seconds. Next, the subject inspires against the gauge, taking care that suction is produced by chest and diaphragm rather than by the buccal muscles, maintaining a negative pressure of about one-half pound for ten seconds. The stop-cock to the spirometer is then opened and the subject blows to his full vital capacity, at least three times in succession, with intervals of three or four seconds. He is requested to expire at a fairly rapid rate, allowing from twelve to fifteen seconds to complete the expiration. One-half minute after the last expiration, a final notation of pressure, pulse rate and rhythm and physical signs is made.

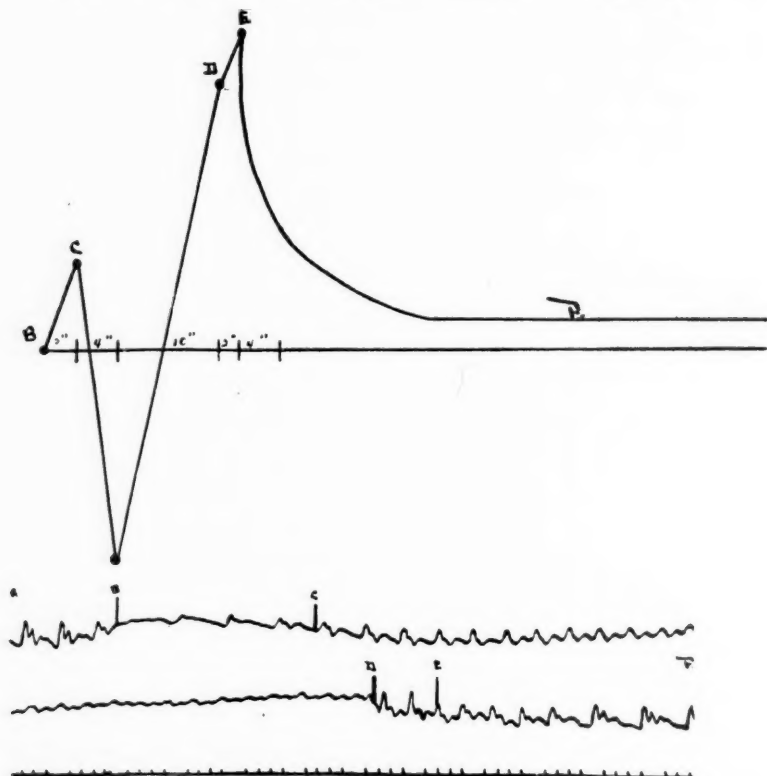
Thus the technique is divided into nine determinations; (1) initial notations of blood pressure, pulse rate and rhythm, cardiac physical signs; (2) full inspiration held; (3) full expiration held; (4) one pound positive pressure; (5) one-half pound negative pressure; (6), (7), (8), expiration to full vital capacity; (9) terminal notation of pressure, pulse rate and rhythm, and cardiac physical signs. Before each of the Steps Two to Eight the systolic pressure is determined, thus permitting an accurate observation of the absolute pressure deviation for each step.

The results are plotted as a curve. In red ink, the systolic pressure noted before each step is undertaken is plotted. This we call the "base-line blood pressure curve." In black ink, the maximum of deviation either above or below the base-line pressure, as the subject completes each step, is plotted. We call this the "reaction curve." Of course in the initial and final determinations, the curves are superimposed.

We have subjected to this test one hundred individuals, male and female, who, in so far as we can determine by the usual methods of physical examination, have normal cardiovascular systems. Without exception, in every case where we were satisfied that the subject was properly performing the test, there has been a similar reaction to each of the steps. The degree of reaction, as would be expected, has varied with the

CHART 2

Steps Six, Seven, Eight - (Expiration to full vital capacity)



Showing fluctuation of systolic pressure in Steps Six to Eight. Point of maximum rise at E taken for plotting reaction curve. Expiration begins at B; a short initial rise B - C; a sharp extensive decline followed by a rise to point D where expiration ceases. Immediately there is a slight, further, rise D - E; thereupon the pressure drops rapidly to a point about 10 mm above the base line, then falls gradually.

The pulse tracing: A - B before expiration; B, beginning expiration; B - C, slowing of pulse synchronous with initial rise of pressure; C - D, pulse increases in rate and decreases in amplitude while the pressure falls and rises; D, expiration ceases; D - E, increased amplitude of pulse corresponding with terminal rise of pressure; E - F, the pulse regains its former amplitude with a slower rate.

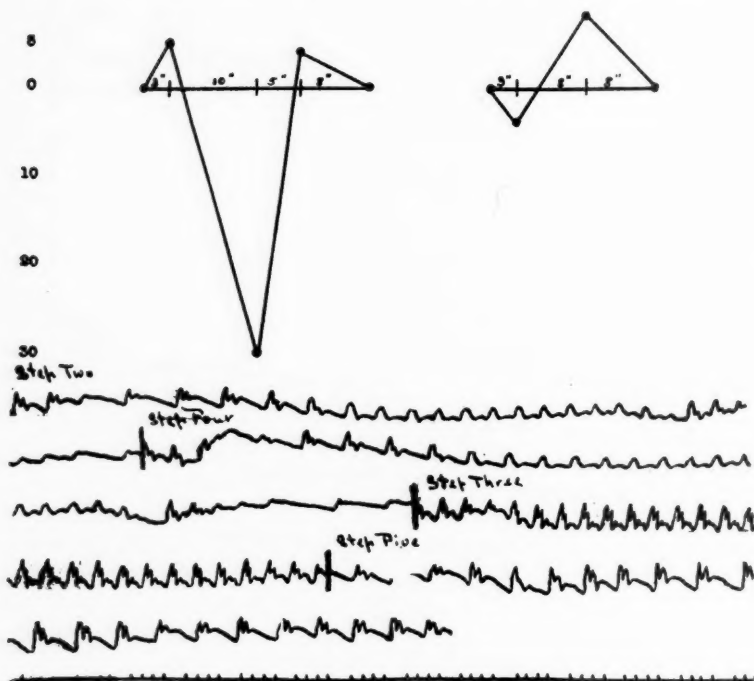
CHART 1

Steps two, four

- (2) (Full Inspiration, held)
(4) (Expiration against gauge)

Steps three, five

- (3) (Full Expiration, held)
(5) (Inspiration against gauge)



Showing fluctuation of systolic pressure in Steps Two - Five. Pulse tracings in these Steps are given below. In Steps Two and Four there is an initial slowing of the pulse, corresponding with rise in pressure, followed by gradual increase in rate and decrease in amplitude until the Step is finished. Immediately the amplitude increases and the rate becomes slower. In Steps Three and Five there is a marked increase in amplitude with gradual slowing of rate to the end of the Step.

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physical structure and the condition of the subjects, the deviation being greater in the larger, more robust, athletic individuals and in the males.

In Step Two, full inspiration held, there is an initial rise of systolic pressure, averaging 5 mm. Hg., of 3-4 seconds' duration, then a sharp decline of 20-50 mm. Hg., frequently approximating the diastolic pressure. As the breath is released, the pressure rises promptly to a point 3-5 mm. Hg. above the base-line, and then gradually declines to normal. In plotting the reaction, the point of maximum decline is chosen. (Chart 1.)

In Step Three, full expiration held, there is an initial decline of 3-5 mm. Hg., for about 5 seconds, then a gradual rise to a point 5-10 mm. Hg. above the base-line. As inspiration begins, the pressure gradually declines to the base-line. The point of maximum rise is plotted. (Chart 1.)

In Step Four, one pound positive pressure held, there is a sharp initial rise of pressure from 5-10 mm. Hg., for about 5 seconds, then a sharp decline of 30-50 mm. Hg. As the intrapulmonary pressure is released, the systolic pressure rises above the base-line. This reaction is similar to that in Step Two except that it is more rapid and greater in degree. The point of maximum decline is plotted. (Chart 1.)

In Step Five, one-half pound negative pressure held, there is an initial fall in pressure, then a gradual rise above the base-line, and subsequent decline when suction is discontinued. This reaction is quite similar to that of Step Three and practically no greater in degree. The point of maximum rise is plotted. (Chart 1.)

In the three succeeding steps, expiration to full vital capacity, there is a sharp initial rise of 5-10 mm. Hg., a sharp decline to a point 20-50 mm. Hg. below the base-line, a gradual rise to a point 20-40 mm. Hg. above the base-line. After the expiration is finished, there is usually a further rise of 5-10 mm. Hg. followed by a fairly rapid decline to a point about 10 mm. Hg. above the base-line and then a gradual fall to the base-line. The point of maximum rise is plotted. (Chart 2.)

We have found that the final blood pressure, both systolic and diastolic taken one-half minute after the conclusion of the test, while occasionally not much altered, is as a rule raised from 5-10 mm. Hg. The pulse rate is generally increased from 5-10 beats per minute. In individuals of athletic habits and in good physical condition, it is frequently diminished.

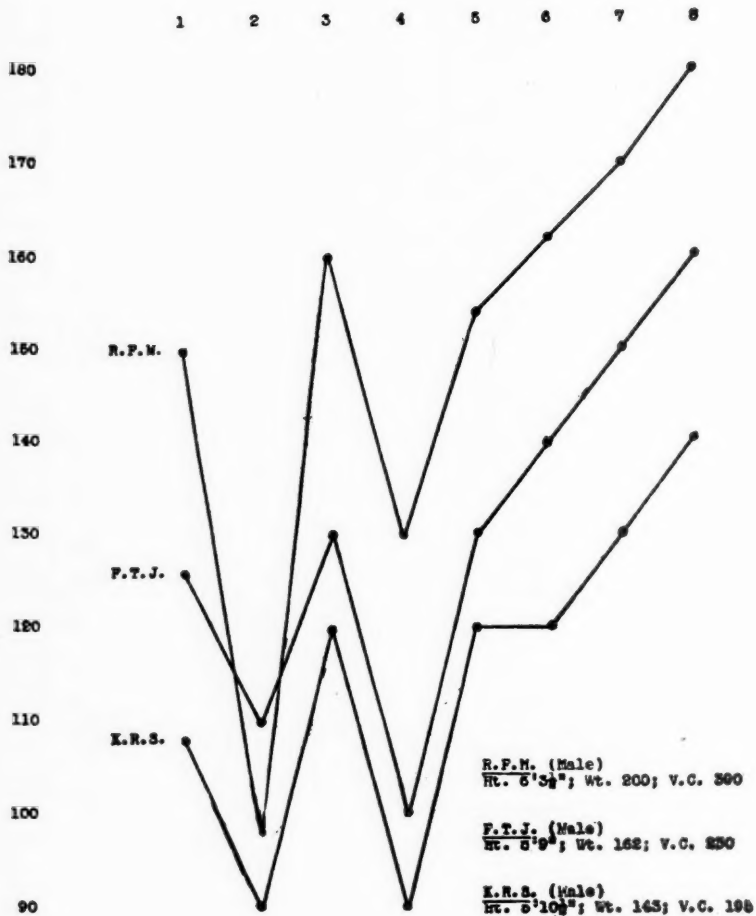
We present here typical charts of the reactions which we have obtained. (Charts 3, 4.) We have made no attempt to plot the change of pulse rate, believing it would unnecessarily complicate the test. We insist, however, upon noting any irregularities of rhythm and tone and any striking change in rate. In a rough way, we have satisfied ourselves that the diastolic pressure varies directly in proportion to the systolic, but to a far lesser degree. Obviously, it is not possible to follow the deviation of diastolic pressure by the auscultatory method with any degree of certitude.

We will now give a summary of our opinion as to the physiological cause of these reactions. We believe it is largely the effect of variations in the amount of blood supplied to the heart which result from the frequent and extensive changes in intrapulmonary, and consequently in intrathoracic, pressure.

Arterial pressure is dependent upon several factors: (1) the force of the heart contractions; (2) the resistance to the arterial stream which results from the friction of lateral pressure and from the small caliber of the arterioles, capillaries and venules; (3) elasticity and tonicity of the arteries; (4) volume of blood in the system. In addition, of course, there are the hydrostatic influences and the changes arising from muscular movements.

It is believed that the venous pressure decreases from the periphery toward the heart, so that in the mediastinum it is practically the same or possibly slightly less than the atmospheric pressure. The intrathoracic pressure, comprising that of the pleural cavity and the mediastinal space, is normally always negative or less than one atmosphere. It equals the intrapulmonary pressure minus the force of elastic recoil of the lungs. It is obvious that by attempting forcibly to expel in-

CHART 3



Reaction Curves of Three Normal Individuals

trapulmonary air the intrathoracic pressure will be increased.

It is possible for the average individual to increase the intrapulmonary pressure by about 100 mm. Hg. This, of course, means that the intrathoracic pressure will likewise be increased to an amount only slightly less. Obviously this will result in temporary collapse of the venæ cavæ, with cessation of the venous flow to the right heart and pulmonary system. As the pulmonary system becomes emptied of its blood, the flow to the left heart will be cut off. This means a decrease in the volume of blood expelled into the arterial system and a fall of pressure.

It is likewise possible for an average individual to decrease the intrapulmonary pressure and consequently the intrathoracic pressure by at least 50 mm. Hg. This obviously would result in an increased supply of blood to the heart and an increased volume expelled into the arterial system.

In Step One, a marked increase in intrathoracic pressure is obtained by the pressure of a relaxed diaphragm and chest upon the air in the expanded lungs, the escape of which is prevented by the closed glottis. In Step Four, the same result is accomplished by forcible expiration against the gauge. Immediately the venous flow to the right heart is cut off. At the same time, the blood in the pulmonary system is subjected to increased pressure tending to drive it forcibly to the left heart. There follows an increased volume of blood to and from the left heart with an increased arterial pressure. This is only temporary, ceasing as soon as the blood in the pulmonary system has been practically expelled. Thereupon follows a fall in arterial pressure for the reason that the blood is cut off from the heart. This will continue until the intrathoracic positive pressure is released or until the venous pressure in the mediastinum has been raised to such an extent that it will overcome the positive intrathoracic pressure.

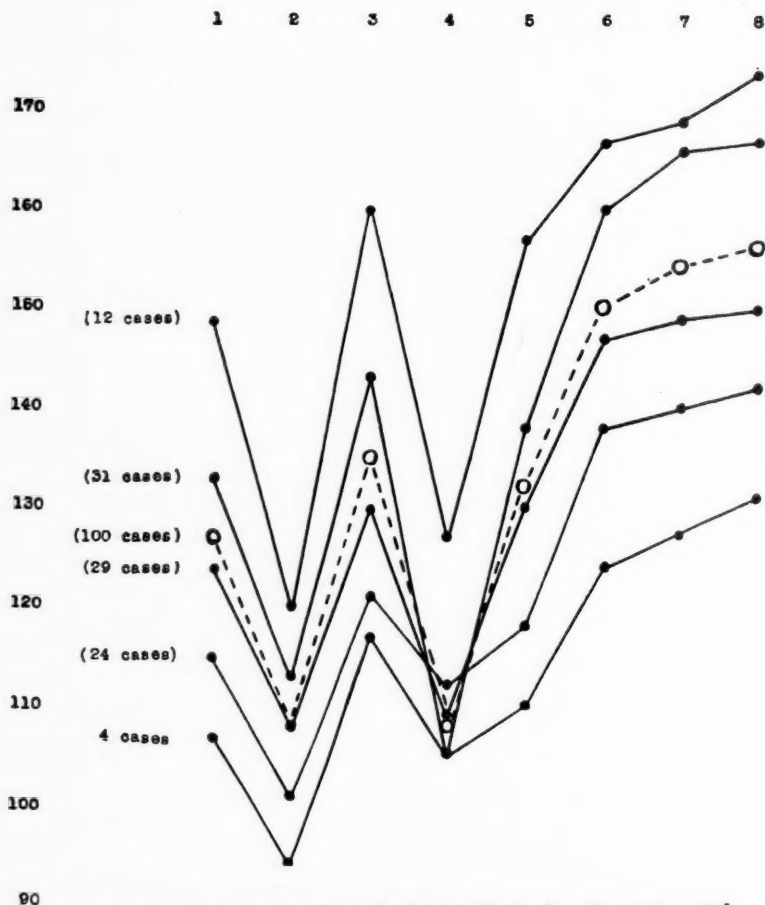
In Steps Three and Five, an opposite effect is obtained. With the decrease in intrathoracic pressure, there is an increased flow of blood to the right heart and at the same time

temporary engorgement of the pulmonary system with diminished flow to the left heart. A temporary decrease in volume expelled by the left heart results in a corresponding fall in arterial pressure. Shortly, however, the increased volume expelled by the right heart overcomes the pulmonary engorgement, with re-establishment of the supply and output of the left heart and increase in arterial pressure.

In the last three steps, expiration to full vital capacity, there is at first a marked increase in intrathoracic pressure. As before explained, this results in immediate forcible expulsion of blood from the pulmonary system and a rise in arterial pressure. The blood to the right heart being cut off, there next follows a fall in arterial pressure. Thereupon, as the venous pressure rises, undoubtedly accelerated by the increased intra-abdominal pressure upon the splanchnic system due to the contraction of diaphragm and abdominal muscles during the forcible expiration, there is gradual restoration of the blood supply to the heart and the arterial pressure begins to rise. As the forcible expiration approaches its conclusion and the lungs become more empty, the intrathoracic pressure gradually becomes less. The venous pressure, however, still remains increased on account of the continued intra-abdominal pressure and thus probably the increased venous supply to the heart is maintained. The heart contractions, reacting to the exertion of blowing, become more rapid and powerful. In addition, there is doubtless reflex reaction of the cardiovascular system to the temporary asphyxia resulting from the previous fall in pressure. From these various factors follows a considerable rise of pressure above the base-line, which continues for a few seconds after expiration has ceased.

If the subject expires at a rapid, rather than a moderate, rate, the intrathoracic pressure is maintained at a high level. This tends to prolong the repression of supply and output of the heart with lowered systolic pressure. Promptly, with the cessation of expiration and decrease of intrathoracic pressure, the blood from the engorged veins pours into the heart, in quantities far greater than normal, and the pressure rises

CHART 4



The reaction curves of one hundred individuals, who were normal, in so far as the usual methods of physical examination could determine. The interrupted curve is the average for the total. The reactions were subdivided into five classes; those with original pressures of, 100-109 mm., 110-119 mm., 120-129 mm., 130-139 mm., 140 mm plus. The conformity of the subdivision reactions with the average is apparent. As a working standard, we consider the average curve above to be the normal.

rapidly to a high level. As stated before, the subject is required to take about fifteen seconds for a complete expiration which we consider a moderate rate.

We have demonstrated to our own satisfaction that the systolic pressure may undergo extreme fluctuations with the changes of intrathoracic pressure which this test produces. In Chart 3 are given three reactions of individuals with original pressures of 150, 126 and 108 mm. Hg. These we believe to be typical reactions. In Chart 4 is given the average curve of the 100 normal cases. These are further subdivided into five groups: those with original pressures from (1) 100 to 109 mm. Hg.; (2) 110 to 119 mm. Hg.; (3) 120 to 129 mm. Hg.; (4) 130 to 139 mm. Hg.; (5) 140 + mm. Hg. The similarity of the subdivision curves with the average is striking.

Of course, it is obvious that one hundred cases is a small group from which to make definite deductions. In view, however, of the similarity of the subdivision and the average curves as shown in Chart 4, we, at present, consider the average curve as the normal and expect the curves of normal cases to conform with it.

We have found the test easy of application, practically all of the subjects performing correctly with the exception of Steps Three and Five. In Step Three many are unable to close the glottis after complete expiration and allow the diaphragm and chest muscles to relax. In Step Five it is often difficult to teach the subject to produce suction with the chest and diaphragm rather than with the buccal muscles. Consequently, in both of these steps it may be difficult to obtain the negative intrapulmonary pressure which is desired. However, the fluctuation of pressure in these steps is but slight and it is probable that they are of little consequence. It will, perhaps, be found advisable to eliminate them.

Occasionally it is difficult to teach the subject to perform Step Two correctly. As explained before, it is necessary after full inspiration to close the glottis and allow the diaphragm and chest muscles to relax. This produces an increase of intrapulmonary pressure. If, however, the subject maintains full

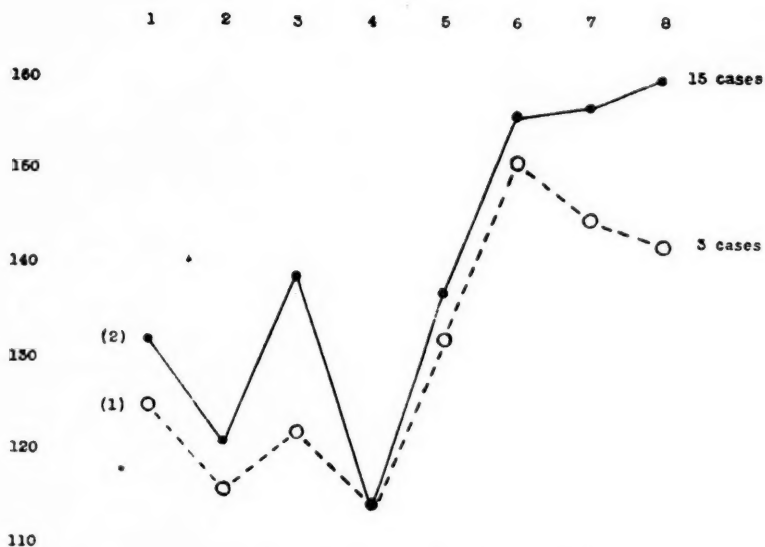
inspiration by a continued chest expansion and contraction of the diaphragm, it is obvious that the intrapulmonary pressure will be practically one atmosphere, which will produce no reaction in blood pressure. Even if the applicant is not able to perform this step properly, the desired increase in intrapulmonary pressure may be obtained invariably in Step Four, expiration against the gauge.

With a little practice, it is easy to follow the systolic pressure in the desired direction. It is not necessary to follow it through all its fluctuations. For instance, in the last three steps, the examiner knows that the pressure will rise temporarily, will next take a sharp drop and will then gradually rise above the original pressure. As it is the final rise which he wishes to obtain, he does not attempt to follow the preceding drop but maintains the pressure in the cuff at a point 15 to 20 mm. above the original pressure and waits until the pressure reaches this point in its upward course. He is then able to follow it easily to its peak. The same holds true for the steps in which the pressure is expected to show a sharp decline.

In the three final steps, blowing through the spirometer to vital capacity, it is necessary that the subject blow not only at a moderate rate but so far as possible at the same rate for the three final reactions. It is likewise necessary that he blow approximately the same amount. We have found that if he blows more in one step than in the other or takes longer for the expiration that the pressure will rise higher. If, moreover, he blows at a very rapid rate, the pressure will remain depressed for a longer period and will not subsequently rise to such a high level.

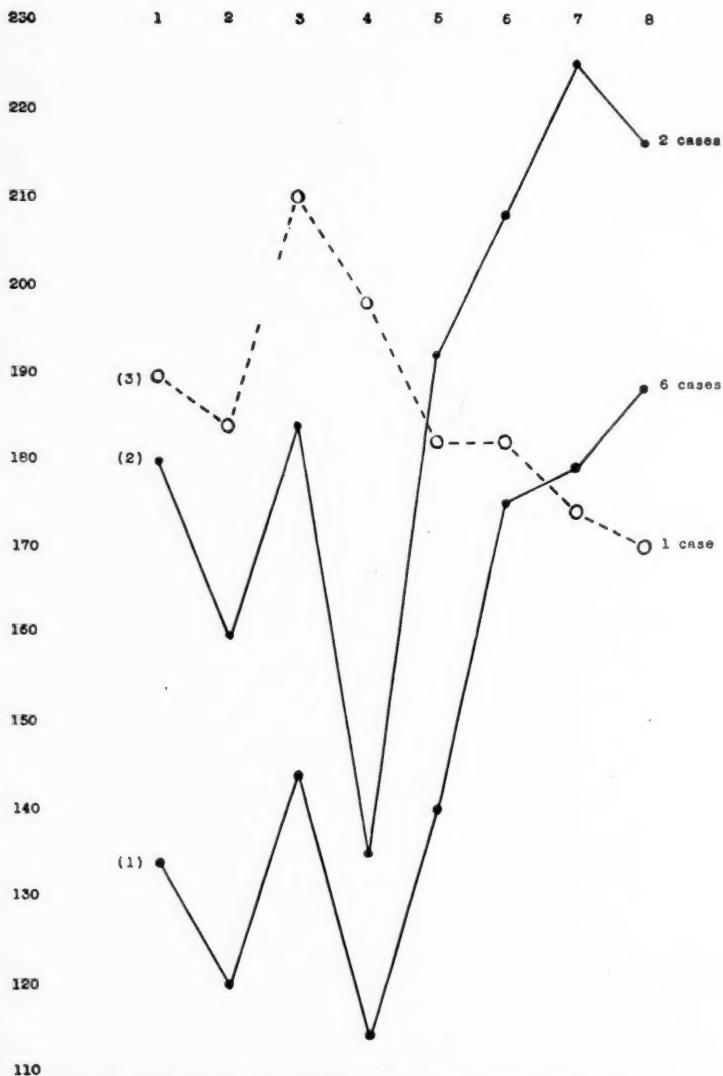
We have subjected to this test 28 cases which, on physical examination, have shown abnormal cardiac sounds. The curves of these cases are given in Charts 5, 6, 7. In Chart 5 are the curves of 18 cases which presented systolic murmur at base or apex. In Curve One, comprising 3 cases, the slight degree of fluctuation in Steps Two, Three and Four and the falling off of pressure in Steps Seven and Eight are apparent. Curve Two conforms with what we consider the normal. In

CHART 5



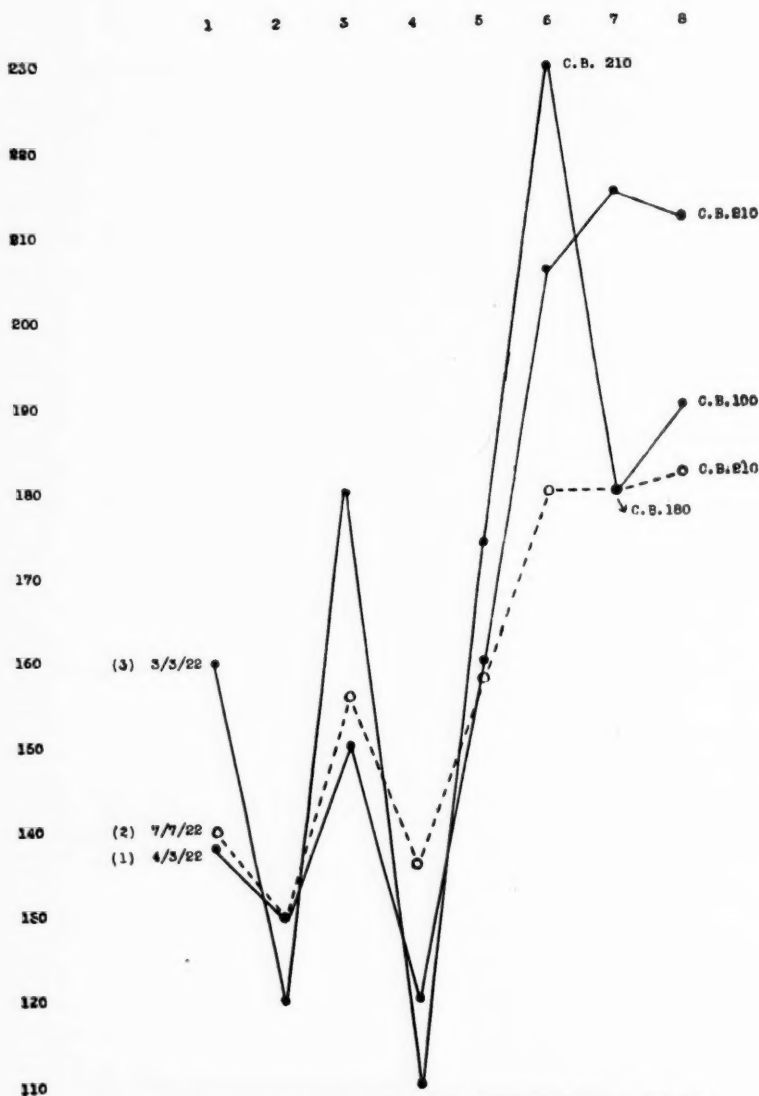
Curve One gives the average reaction of three cases of systolic murmur. This reaction is atypical, showing a restricted degree of fluctuation and a terminal fall of pressure. Curve Two gives the average reaction of fifteen cases of systolic murmur which conforms with the normal.

CHART 6



Curve One gives the average reaction of six cases of hypertrophy; Curve Two, of two cases of hypertension without demonstrable hypertrophy. Both these conform with the normal, except for a wider fluctuation of pressure, particularly in the last two steps. Curve Three gives the reaction in a case of hypertension with hypertrophy. It is atypical, with a striking fall of pressure at the final steps.

CHART 7



This applicant, when examined on March 5rd, presented a heart slightly enlarged to percussion, a systolic murmur at the apex, and the reaction of Curve Three. The wide variation of systolic pressure is striking. In Step Six it reached 230 mm. Hg. In Steps Seven and Eight it was deemed inadvisable to push the pressure so far; the capacity required was reduced. Curve One gives the reaction one month later; Curve Two, four months later. This conforms with the normal. The abnormal physical signs had disappeared, under treatment. C. B. indicates the capacity in cubic inches blown through the spirometer.

Chart 6 are plotted the curves of 9 cases of hypertension and hypertrophy. In Curve One, comprising 6 cases of hypertrophy, we note that the pressure fluctuation is greater than in what we consider the normal curve. In Curve Two, comprising 2 cases of hypertension without demonstrable hypertrophy, we note likewise the greater fluctuation of systolic pressure. Both these curves conform, excepting the extent of fluctuation, with our conception of the normal. Curve Three represents the reaction of a case of hypertension and hypertrophy which is not only atypical but shows a definite falling off of blood pressure in the final steps.

In Chart 7 are shown three reactions of a case originally diagnosed as hypertrophy, with a systolic murmur at the apex. The reaction in Curve Three reveals an unusually wide fluctuation of systolic pressure. In Step Six this rose to 230 mm. Hg. In the next two steps the subject was not permitted to blow to so great a capacity, with the result that the systolic pressure did not rise as high. Curve One represents the reaction one month later. During the interval the subject had carefully followed instructions as to the regulation of his habits, the amount of work, sleep, exercise, etc. At this examination, the systolic murmur was much less apparent. There was no demonstrable enlargement of the heart. Curve Two represents the final reaction four months after the original examination. At this time, the systolic murmur had entirely disappeared and no enlargement of the heart could be demonstrated. It will be noted that this curve conforms with the normal.

We have not the least desire to force any conclusions from the small number of cases which have been tested. As a working standard, we consider that reactions which show a definite decline of systolic pressure in the last two steps, as in Curve One, Chart 5, and Curve Three, Chart 6, are abnormal. Inasmuch as the rise of systolic pressure in these steps is probably due in the main to the power of the heart muscle, we feel that inability to maintain the pressure attained in Step Six, providing always that the subject blows at the same rate and to the same capacity, may indicate weakened heart muscle. On

the other hand, if the systolic pressure rises in the two final steps to a level obviously higher than the normal, we feel that this may be an indication of a heart muscle unusually powerful: in other words, hypertrophied.

In three cases we have noted precordial pain, irregularity of rhythm and tonal arrhythmia. These were not apparent before the test but developed while it was being performed. We consider these to be signs of abnormality.

Chart 8 is a specimen of the chart now in use in the Medical Department at the Home Office of the New England Mutual Life Insurance Company. These are filed away for future reference with the examination papers of the applicant.

In conclusion, we wish to emphasize again that we consider this investigation, as yet, purely experimental. We present it as an interesting and, so far as we have been able to determine from examination of the literature, a unique study of the reaction of the cardio-vascular system to rapid and extreme changes of intrathoracic pressure. The limited application to abnormal cases, while suggestive, does not warrant the claim that this is a real test of cardio-vascular function. It is our intention to continue the investigation, particularly with applicants presenting abnormal cardiac conditions.

BIBLIOGRAPHY

1. CHRIST. *Archiv. f. klin. Med.*, 1894, liii, 1902.
2. SELIG. *Prag. Med. Wchnschr.*, 1905, xxx, 418; 1905, xxxi, 432.
3. GRAUPNER. *Berl. klin. Wchnschr.*, 1902, 174; *Deutsch. Med. Wchnschr.*, 1906, xxxii, 1029.
4. MENDELSON. *XIX Kongr. f. Ind. Med.*, 1901.
5. BARRINGER. *Archives of Int. Med.*, vol. xvii, 363.
6. LIAN. *Ann. de Méd.*, Paris, 1918, v, 345.
7. KAHN. *Functional Diagnosis*, 1920, 158.
8. HERZ. *Deutsch. Med. Wchnschr.*, 1905, xxxi, 215.
9. HERZ. *Lehrbuch der Heilgymnastik*, Wien, 1904.
10. KATZENSTEIN. *Deutsch. Med. Wchnschr.*, 1904, xxx, 807; 1915, xl, 457.
11. *Die Herz Krankheiten*, Wien, 1912, 125.
12. COOPER. *J. A. M. A.*, 1909, lii, 1182.
13. ABRAMS. *N. Y. Med. Jour.*, Oct. 29, 1910, xcii, 850.

CHART 8

Cardio-Respiratory Test

John Doe

March 27, 1922

Before Test:

S - 120
D - 80
P - 72

After Test:

S - 128
D - 86
P - 72

V.C. 220

C.B. 220

(or)

C.B. (1) 220

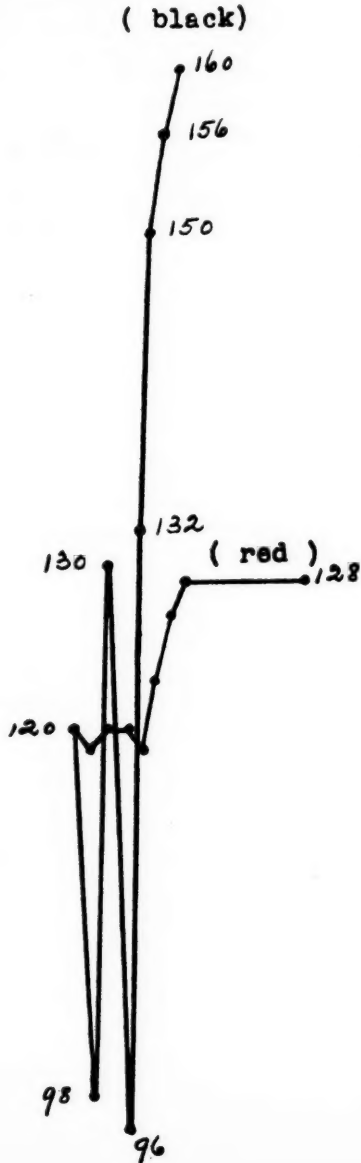
(2) 200

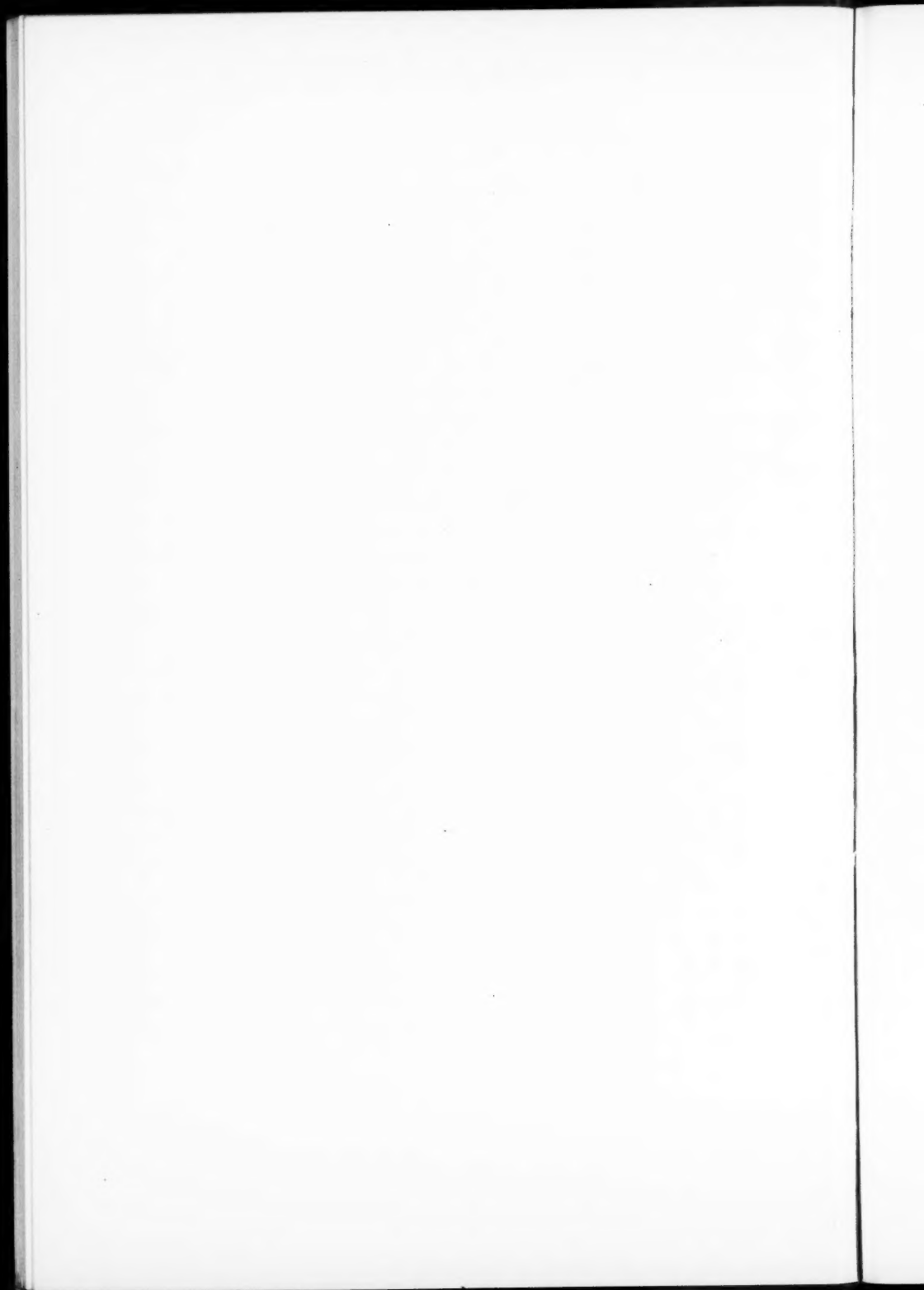
(3) 180

(A specimen Chart, as used at Home Office of
New England Mutual Life Insurance Company)

Signature of Examiner.

Cardiac physical signs noted here.
Examiner's opinion of normality of reaction





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14. MERKLEN AND HERTZ. *Examen et séméiotique du cœur*, Paris, 1910.
15. MINERBI. *Riv. crit. di clin. med.*, Florence, 1917, xviii, 213.
16. BRECCIA. *Riforma Med.*, Naples, 1917, xxxiii, 1133.
17. KORANYI. *Ztschr. f. klin. Med.*, 1897, xxxiii, I.
18. LOEB AND KNECHT. *Deutsch. Arch. f. klin. Med.*, 1905, lxxxiii, 462.
19. TIEGERSTEDT. *Ergebn. d. Physiol.*, 1907, vi, 265
20. GOODMAN AND HOWELL. *Am. Jour. Med. Sc.*, 1911, cxlii, 334.
21. STONE. *J. A. M. A.*, 1913, lxi, 1256.

Dr. McMahon—The subject is open for discussion and I am sure Dr. Frost will be very glad to answer any questions that may suggest themselves.

Dr. Exton—The Association certainly owes a debt of gratitude to the New England Mutual for the work they have done on this important problem. What we need more than anything else are measuring rods for the different conditions which may be suspected in our applicants and of these none is more often in question than the fitness of the heart muscle. From a physiological standpoint this is certainly an extremely interesting work and much too complicated a test to attempt to evaluate at the present time. The point is that Life Insurance men need to do just such work as this in order to justify their existence, and we owe Dr. Dwight and Dr. Frost every encouragement to go on with their experimental work.

In view of the fact that the X-Ray has taught us that the older methods of physical diagnosis often do not give even reasonably accurate results in determining degrees of hypertrophy it may be suggested that cases which are studied from the standpoint of hypertrophy might with advantage be checked up with the X-Ray. By the use of the X-Ray factors which may not show up on physical examination, such as abnormalities in the arch of the aorta and the pulmonary artery may be found which should always be reckoned with in considerations involving the effects of intra-thoracic pressure.

I would like to ask Dr. Frost regarding Chart 7, if I understood him correctly the records were made of the same individual after a period of four months had elapsed, and that

this man had hypertrophy on the first examination and gave an abnormal record, but that on the last test the records appear normal and the physical signs of hypertrophy had disappeared. Is this to be taken as an indication of improvement in this man's condition?

Dr. Frost—That is what we believe.

Dr. Exton—That is to me a most interesting demonstration. In the cases that you have regarded as normal have you checked these up by making repeated readings?

Dr. Frost—We have not.

Dr. Exton—I think that would be a very good plan. I can see that the rise and fall of the first stage probably does not give the best indication as to the meaning given by the test. Please do not think that I am criticizing in any way, because I do feel that no one could reasonably gauge so complex a matter off-hand well enough to justify criticism. I am trying to do what I can to encourage because I think it is well worth while and very promising, but I would like to suggest that in connection with tonal arrhythmias or marked variations of pressure the question of pulmonary conditions such as emphysema, etc., might enter. Even the amount of gas in the stomach may be relevant. Clinically it is well known that even severe arrhythmias occur as the result of gas in the intestinal tract.

It seems to me that the terminal stages of this test have greater possibilities than the primary, and I am inclined to think that the closure of the glottis will be found somewhat difficult to teach some individuals and that possibly that part of the reaction could be omitted as not being as valuable as the two terminal stages, although it appears likely that nervous reactions will influence these.

I congratulate Dr. Dwight and Dr. Frost on this work and hope that they will go on with it and succeed in evaluating the effects of the different factors which are necessarily involved in the reaction.

Dr. Symonds—I would like to ask Dr. Frost whether they have made any experiments in cases of fibrillation.

Dr. Pauli—This is rather an ingenious and original paper

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and we seldom have a paper presented before the Association like this one. It rather reminds me of the days of 1907 when Dr. Rogers presented his blood pressure instrument.

The essayist has permitted me to read this paper this morning before the meeting and has asked me to say a few words regarding the physiology or the application of physiology to the paper. I will admit that I do not feel qualified to discuss this phase of the subject and I feel that no man excepting perhaps Dr. Joseph Erlanger of Washington University of St. Louis, who is in touch with this class of work, should undertake it, but I will be glad to express my opinion regarding the test as it appeals to me at the present time, without a great deal of consideration.

When we consider such a functional test of the heart as increased thoracic pressure, it is essential that we should consider two points, first the physiology of the lungs and cardiovascular system, and second, the physics of blood pressure.

The essayist has explained that there are four essential points in any problem of blood pressure, first, the pump; second, the amount of fluid; third, the condition of the tone or elasticity of the vessels; and fourth, peripheral resistance. Now if you affect one of these factors you affect the blood pressure; if you have a stronger pump you increase it, and weaker, you diminish it; increase your resistance, you increase your blood pressure; if your capillaries are dilated you diminish your blood pressure.

Now what effect has the respiration on the blood pressure? Let me remind you of the days when you were working in the physiological laboratory, and you performed your blood pressure experiments on the dog, and placed a canula in carotid artery, connected it with a mercury manometer and recorded the blood pressure tracing on the kymograph. You will recall that the pulse tracing was not on a straight line parallel with your base line. The pulse tracing was a waving line with a gradual rise and fall in blood pressure, so-called Tauber-Hering curves, indicating a rhythmic action of the vaso-motor center. Then, too, during inspiration the blood pressure

curve rises, and in expiration the curve falls, due to the suction action of chest cavity during inspiration and increased intrathoracic pressure during expiration.

Now then, let me also go into the vaso-motor effect in connection with blood pressure. In your blood pressure experiment on the dog, when you stimulated the vagus nerve with electric current from the induction coil, the blood pressure dropped nearly to the base line, due to inhibitory action on the heart, and then gradually rose to the normal blood pressure level. You also demonstrated the effect of stimulating the sympathetic or vasoconstrictor nerve to the splanchnic area; your blood pressure was increased from constriction of vessels in the great splanchnic tank and the curve gradually rose to a higher level and then returned to normal level after the stimulation was removed.

I mention these experiments to emphasize the importance of the vaso-motor nervous system in considering a blood pressure test as a functional heart test, especially in relation to the effect of intra-thoracic pressure.

It is very important in considering the blood pressure as a functional test to keep in mind your vaso-motor control.

The important point in the curves which Dr. Frost demonstrated is the fourth, fifth and sixth phase, whether the heart can sustain that increased pressure. The essayist draws the conclusion that if the heart is hypertrophied it will give the curve with increased pressure; if the heart muscle is weak, the blood pressure is not sustained and the curve falls below the normal. Is the sixth phase of the test due to the heart action or to vaso-motor control? That is a very important point in relation to the test and I am not prepared to answer.

Let us go a little into the explanation which the essayist has taken up. There is one point that I think deserves a little further consideration, that is, that when the intra-thoracic pressure is increased, he believes that the venæ cavæ become occluded, and there is no blood transmitted to the heart. Now the heart and blood vessels are in the mediastinum, the thoracic cavity is limited by the pleura. There is some ques-

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tion in my mind whether the increased intra-thoracic pressure will affect the vena cavæ to the heart, for this reason: that when you had the dog on the table and you resected a rib and then another rib on the other side and used your bellows to continue the respiration you permitted one atmospheric pressure to enter the thoracic cavity, a pressure of about fifteen pounds. Now what effect did that have on your blood pressure? It did not occlude the vena cavæ of the heart, and therefore, I think that point will deserve a little further experimentation in order to verify such a conclusion.

I will admit, however, that such experiment is not analogous with the experiment as conducted in the human being because in the human being you have a voluntary forced expiration.

I feel that I would like to have an opportunity to work with this instrument. It has wonderful possibilities and I am inclined to think that in time we will be able to make use of it, especially the fourth, fifth and sixth phase of these reactions. I want to congratulate the essayist on the amount of work he has done and the rather ingenious paper which he has presented to this Association.

Dr. Gordon Wilson—I want to take exception to one remark, and that is regarding the contribution of this Association to science. I do not know of any association in this country that has contributed more. The Medico-Actuarial report alone is a sufficient contribution to justify this Association's existence, and its importance will be more and more appreciated. It is a great regret to me that a copy of this report is not in the library of every medical school and the larger medical libraries in this country. Another contribution by a member of this Association is the Tycos sphygmomanometer, which has so popularized the taking of blood pressure that there has been accumulated a mass of data that has certainly broadened the general knowledge of cardiac diseases, and has made possible extremely valuable statistical studies.

This instrument has given us an easy clinical method of recognizing even mild variations of blood pressure as seen in "pulsus alternans" and "pulsus paradoxus," especially the

cases of the latter as seen in those suffering from bronchial Asthma and Emphysema, and the test reported to-day is based upon the same factor that causes "pulsus paradoxus"; namely, the variations of blood pressure due to variations in intra-thoracic pressure.

I would like to ask the question as to the variation of the pulse-rate in these cases, because that would have a bearing on the question of systolic pressure.

This contribution, to my mind, gives promise of being the most valuable that I have ever listened to at any meeting of any medical association.

Dr. Archibald—I would like to ask one question about Chart 6. It strikes me as possible that that particular individual may have become somewhat nervous, may have had a stimulation of the splanchnic area, which might have accounted for his fagging out during the test. We see this under ordinary conditions very often in making blood pressure tests—sometimes one or two tests have to be made in order to get an accurate determination, because the patient becomes nervous.

Dr. Porter—I want to express my personal thanks for this contribution because if it proves satisfactory it will fill a decided need. Our present methods are most unsatisfactory. There is nothing scientific in having a man hop on one foot, nor is there anything uniform in such method, and we need just this particular test. I shall watch the results with great interest, and I hope for an opportunity for personal work along similar lines.

I would like to ask Dr. Frost regarding Chart 7, whether the individual was of the type that might possibly have access to an instrument shop during that period, or whether he saw you or any other physician so that he might have experimented—a man who knows how, in other words, can get one kind of findings, and the man who doesn't know cannot get them, and I wonder whether in that period this particular case in Chart 7 had been experimenting at all, because that is a most interesting point.

Dr. Rogers—It looks as if we were really entering into still

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another era of progress in Life Insurance work. Whether this plan, as mapped out by Dr. Dwight and Dr. Frost will stand the test of exhaustive experimentation has yet to be proven, but if it does, and if the technique can be simplified so that we can use it, one of the most important problems in Life Insurance will have been solved, for the reason that we shall be able to recognize in advance what we very much need to recognize, and that is, those hearts that are about to give out. The sphygmomanometer will not give us this, and, as has been said, a gymnastic test of any sort is very unsatisfactory, because it weighs so unevenly on different people. Some people who are accustomed to plenty of exercise respond very little to that test, and others who take very little exercise show a considerable response to it. As I understand it from Dr. Dwight, this test will try out any heart, whether it is a heart that is accustomed to certain exertions or not. It looks as if, through the efforts of these gentlemen, we are going to have something of great value to all of us. I also want to thank both of them.

Dr. Frost—Mr. President and Gentlemen: In regard to the question of emphysema, I do not know.

With regard to arrhythmia and gas in the stomach, it seems to me probable to suppose that an individual who at the beginning of the test did not show a tonal arrhythmia would not show it throughout the test if he were normal—if he had gas in his stomach at the beginning of the test I assume that he would have an arrhythmia then.

In regard to Dr. Symonds' query as to fibrillation, we have not tried this out on fibrillation cases or any of that type of cases. It is logical that in the development of the test it will have to be carried out in such types of cases if we are to come to a real estimate as to its value. We hope in the future that we can do this.

With regard to Dr. Pauli's discussion, I regret extremely that, from a sense of modesty perhaps, being unwilling to bore you with what seemed to me to be dry physiology, I did not give my conception as to the physiological cause of these reactions, although you will find in my manuscript my opinion in

detail. Dr. Pauli made the statement that on rib resection with the entrance of air through the pleural cavity, there is an immediate increase in intra-thoracic pressure of one atmosphere. That, I believe, to be incorrect. Atmospheric pressure, so the physiologists tell us, and as I conceive it, is equal to intra-pulmonary pressure minus the force of elastic recoil of the lung. Now intra-pulmonary pressure of course varies four to five mms. with inspiration and expiration above and below one atmosphere. With the chest fixed, the glottis open, the intra-pulmonary pressure is one atmosphere or 760 mms. of mercury. At that time the intra-thoracic pressure will be 760 mms. of mercury minus the force of the elastic recoil of the lungs, which in full inspiration is about 7 mms. of mercury and on full expiration a little over four mms. of mercury. Therefore regardless of the amount of pressure you have in your lungs your intra-thoracic pressure will never vary more than 8 mms. from the intra-pulmonary pressure. Therefore I believe that this criticism as to the effects of rib resection, producing greatly increased intra-thoracic pressure, is incorrect.

Dr. Archibald raises the question in Chart 6, whether the subject may not have suffered a vaso-motor collapse such as that often seen in taking blood pressures of apparently normal individuals, when an examiner is accustomed to having an applicant faint occasionally. Of course I cannot say absolutely that this did not happen, but I do not believe it did. There was no variation in the pulse rate and the applicant showed no other signs that you would get ordinarily under those circumstances.

Dr. Porter raises the question of—to put it in my own words—trickery in the reactions obtained in Chart 7. I do not believe that happened.

Dr. Porter—I did not mean, Dr. Frost, to convey the idea of trickery, but there are so many tricks in the way of applying that instrument and I wondered whether that man in the interim had been experimenting himself so as to do away with these extreme findings, rather than any intention to misrepresent himself.

Dr. Frost—I do not believe he could because it is very difficult to falsify the findings.

Dr. Wilson raises the question of the reaction of the pulse during these various steps. I intentionally did not dwell upon that, as the tracings of the reaction of the pulse appear upon the charts which will be published in the "Proceedings."

Regarding the last chart, this is a type we are using now in the New England Mutual. We give in each case the base line and the reaction curves; we give his full vital capacity and the amount he blows for that test, the figures for his systolic, the pulse rate, and we note the physical signs. The examiner writes his opinion of the test and signs it. We file the chart away with the applicant's papers.

I believe I have answered all the questions and I want to thank you for the interest you have manifested.

Dr. Pauli—How many readings were there in Curve 3, Chart 6?

Dr. Frost—One.

Dr. McMahon—Dr. Scadding will now present a report of the work of the Committee of Actuaries and Medical Directors which has been in conference in Toronto for the past two years.

AN ENDEAVOR ON THE PART OF CANADIAN COMPANIES TO SECURE GREATER UNIFORMITY IN THE TREATMENT OF IMPAIRED RISKS.

BY DR. H. C. SCADDING

In Canada for the last three or four years we have annually convened the Medical Directors and Actuaries of those Canadian companies represented in the Life Managers Association for the purpose of discussing ratings for various impairments.

We have been glad to see and to welcome any officers of companies not represented in the Canadian Life Officers Association and officers of any American companies repre-

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sented in that Association, though we felt at the outset in 1919 that we could not ask the latter companies to be guided by any suggested uniformity of practice which the Canadian companies might be willing to follow owing to the small proportion of the business written in Canada to their total American business.

We have been greatly honored by notable representatives of the large American companies at the Annual Meetings and we should like to express our gratitude to them for the great assistance they have given us.

It had long been observed by those of us doing sub-standard business that the widest difference in the treatment of certain classes of impaired lives obtained, and it was with the object of securing a greater weight and, if possible, unanimity of medico-actuarial opinion in dealing with such risks that the officers of these companies have met. We have no organization, that being thought entirely unnecessary in view of our membership in the Association of Life Insurance Medical Directors and the Actuarial Society of America.

That there were differences, to us unexplainable, in the treatment of sub-standard cases existing between the various companies presided over by our American cousins we knew, but we were not blind to our own far greater inconsistencies, and thought it proper to busy ourselves in putting our own house in order instead of speculating on how long it would take them to reach anything like a tidy uniformity.

As insurance officers it was difficult enough to explain to each other the reasons for such flagrant diversity of treatment, to agents it was "a bit thick," and to the ordinary applicant, utterly unintelligible. It was little wonder the insuring public of the intellectually sound, but physically impaired variety, held the haphazard decisions in contempt.

I think we may in all humility congratulate ourselves that for some time we have been at least alive to our own faults and are doing our utmost to resolve the discord of empiricism, and to change the barbaric music of the one-man band to the greater harmony of the balanced symphony.

Time was when some of us thought it presumptive for an

Actuary or other officer to question a medical judgment based upon the experience gained in a practice of five and twenty, or thirty years. Now we rejoice to find our footsteps dogged by any number of these wise counsellors, realizing that the teaching of a single expert, based on as large a personal experience as is possible for one man to accumulate, may prove a broken reed. Apart from occasional actuarial martyrdom in having to listen to lengthy medical disquisitions which are frequently very wide of the mark, our annual conventions have been distinctly interesting.

At the first Annual Meeting a Committee of seven—three medical advisers and four actuaries—was appointed to study the more common and important impairments and suggest a method or methods of treatment. The suggestions were embodied in bulletins and sent to the various member companies from time to time during the year and finally discussed at the succeeding Annual Meeting where certain of the recommendations were adopted and others, after free discussion, referred again to the Committee for modification.

While the resulting recommendations were to be regarded only as a guide to a more uniform practice, it was desirable to follow such as closely as possible through the ensuing year and at the next Annual Meeting free criticism was invited with a view to "change for the better" the aim being to secure as liberal treatment as possible without exceeding the bounds of reasonable safety.

So far our endeavors have produced the happiest results in the field and much benefit to all the Companies from every standpoint.

Very early in these studies the majority of the Committee was converted to the numerical method, but whether that were followed or not, it was felt that the values set forth from time to time would bring us nearer to the goal of more accurate, scientific, and fairer treatment. The colossal stride the medico-actuarial investigation enabled Life Insurance Companies to take is perhaps dwarfed by the inspiration to further effort which that prodigious work created.

Recognizing the worthlessness of individual experience, may we be permitted to refer to an enquiry as touching, if not proving, the value of a consensus of opinions of skilled medical and surgical practitioners untrained in insurance procedure.

At the conclusion of the war it was felt that the Canadian companies at least would probably have to deal with a fair proportion of those who had suffered body mutilation, so one of us set about to obtain an estimate of mortalities to be expected in cases having suffered various war mutilations and he incidentally enquired as to mutilations (if such they may be called for insurance purposes) at the hands of surgeons.

A questionnaire (politely called a guesstionnaire by one of our colleagues) was designed to give the replies as far as possible the necessary homogeneity to enable us to classify the subject matter, together with a letter containing a short paragraph explanatory (as far as could be done in a sentence or two) what extra mortalities meant. About three hundred "Questionnaires" were sent to various physicians and surgeons of proved and acknowledged ability, representing all branches of the Art, and the smallness of the number (53) in replying strictly according to the form, was apparently due to various causes. A goodly number had been called to the Colors and were absent on military duty. Some had joined the great majority. The remainder did not "all with one consent begin to make excuse," and most were kind enough to reply in some fashion. Some were pressed for time, some were engaged in work of a special character and, therefore, could not answer. Others frankly stated that they did not understand the paragraph referring to extra mortality; others evidently did not realize that it was merely a question of "estimating" based on the experience and judgment of the referee. Still others were quite at a loss to know how to answer, saying that they had no data and no knowledge of the subject, and it was felt that some hesitated to put their signatures to anything so frivolous or perhaps important. One personal friend expressed in jest a reluctance that others may have felt by saying that he would

agree to record his guesses provided they would not be held up as evidence against him!

Before the receipt of the replies, there was much misgiving as to the value of the resulting averages of the combined guesses by those utterly unaccustomed to thinking in mortalities. The result amazed us and I think the members of this association will agree that at least a fair number of the medical profession outside our ranks would require very little training to fit them for our exalted and important jobs. Here follows the schedule of average ratings:

53 ANSWERS AVERAGE OF ESTIMATED RATINGS

HEAD

Wounds causing fracture of skull. 139%

CHEST

1. Penetrating bullet wound without infection without hemothorax. 106%
2. Penetrating bullet wound, without infection, with hemothorax. 118%
3. Gunshot wound of the chest followed by hemothorax and infection, and where recovery seems to have followed within the year. 134%
4. Cases requiring operation upon the chest for the relief of empyema, following gunshot wound. 145%

ABDOMEN

1. Cases where operation has been undertaken for the relief of gastric ulcer. 135%
2. Cases where operation has been undertaken for the relief of duodenal ulcer. 133%
3. Cases where operation has been undertaken for the removal of gallstones with drainage of gall bladder. 127%
4. Cases where gall bladder has been removed. 122%
5. Cases of gunshot wound with repair of tears of intestine and infection. 140%

PELVIS

1. Cases where operation has been undertaken for removal of uterus for causes other than malignant disease. 116%
2. Cases of operation for removal of adnexa only. 116%

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Cases where amputation has been performed as the result of trauma:

Below Elbow.....	106%	Below Knee.....	111%
Above Elbow.....	109%	Above Knee.....	122%
At Shoulder.....	119%	At the Hip.....	137%

The particular aim was to secure the profession's average estimate as to the mortalities to be expected in cases having suffered amputation, of which there were a great many, but the gunshot character of the enquiry brought down other averages, which we thought were interesting.

Injuries to the head and chest, Sections 3, 4, and 5, of the abdominal mutilations and pelvic operations, have not yet been considered by the Committee, but the Selecting Committee of my own Company has taken these averages into consideration in dealing with the cases.

AMPUTATIONS

Amputation of the Arm.

These may be taken at standard rates where the amputation has been done at any level other than at or near the shoulder joint (Estimated that a Mortality of 115% exists.)

Provide for a Mortality of at least 135% for those cases that have suffered amputation at or near the shoulder joint or disarticulation of this joint.

Amputation of the Leg.

Below the knee it is suggested that a Mortality of 115% exists. Above the knee and below the junction of the middle and upper third of the thigh—provide for a mortality of 125%.

Amputation near the hip joint or disarticulation at the hip joint—provide for a mortality of at least 135%.

TOTAL DISABILITY in all cases of amputation disallowed.

Amputations, as a result of disease, require special consideration.

The following impairments, listed in alphabetical order, have been dealt with by the Committee and adopted at one or other of the General Meetings.

Scadding—Impaired Risks

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ALBUMEN (WITHOUT CASTS)

History of, or Found on Examination

1. Faint trace
2. Trace
3. Moderate or large

A—Accidental—Once

B—Intermittent—2 out of 3 or 4

C—Constant—3 out of 3 or 4

		Under Age 30	Age 30 to 45	Over Age 45
1.	A B C	S 10 30	S 20 50	S 50 75 up
2.	A B C	S 15 40	S 25 60	S 60 100 up
3.	A B C	S 25 Cannot recommend rating.	15 35	20 75 up

CASTS (WITHOUT ALBUMEN)

(5 Found—Slide)

1.	A B C	S 20 30	S 20 30	S 15 25
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For over 5 found, up to 12 or 15—Slide. Add 10 points.

N.B. 1. On plans terminating at or before age 50, certain individuals of the above classes may be entitled to lesser ratings.

2. Albumen and Casts—Cannot recommend rating.

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APPENDICITIS

Without Removal:

1. POSTPONE for six months from the date of attack.
2. If application between six and twelve months from the date of attack, accept with flat extra of \$7. per thousand per annum for three years.

If application between twelve and eighteen months after date of attack, accept with flat extra of \$6. per thousand per annum, two years.

If application between eighteen months and twenty-four months, accept with flat extra of \$5. per thousand per annum for two years.

If application between twenty-four and thirty-six months from date of attack, accept with single extra of \$5.

After three years from date of attack, STANDARD.

Appendicitis, *with removal*, uncomplicated.

POSTPONE until full recovery (usually at least three months); then STANDARD.

Appendicitis *with Operation for Removal, complicated*.

Cannot recommend rating.

ASTHMA, BRONCHIAL

Last attack within *two years*—plus 7 years, irrespective of age or plan.

Last attack *within two to five years*—plus five years, irrespective of age or plan.

Last attack *over five years*—usually standard.

The above ratings are suggested for mild or moderate types.

For cases of severe type or at advanced ages at entry, cannot recommend rating.

BLIND (*Totally*)

\$3 for Endowment 20 years or less.

\$5 for all other plans.

BLOOD PRESSURE

A number of companies were canvassed and a questionnaire submitted as to the extra mortalities which might be expected in cases where the systolic blood pressure was fifteen or more mm. Hg. above the Hunter-Roger's Table of Averages, Volume 6, page 97, *Transactions, Association Life Insurance Medical Directors of America*. The average systolic blood pressure readings given in Table A and the extra mortality rating given in Table B following are approximately those employed by several of the large American Companies.

TABLE "A"

<i>Age</i>	<i>Average Systolic Blood Pressure</i>
20	122 mm. Hg.
30	124 mm. Hg.
40	127 mm. Hg.
50	133 mm. Hg.
60	134 mm. Hg.

TABLE "B"

<i>Persistent Excess of Systolic Blood Pressure Over Average</i>	<i>Extra Mortality Rating</i>
15 mm. Hg.	10%
20 mm. Hg.	20%
25 mm. Hg.	30%
30 mm. Hg.	50%

In connection with the above, it should be noted that the blood pressures are supposed to be persistently in the neighborhood of the readings shown above, and do not represent some accidental findings.

In general, the companies doing only standard business ordinarily place the danger line at fifteen points above what they consider to be the average for the age (such averages being

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generally those shown in Table "A" above), and they usually decline if the findings are constantly fifteen points in excess of these averages.

DIASTOLIC PRESSURE

Some companies refer to the importance of the diastolic pressure, and would decline if that were constantly over 100 mm. Hg. at any age. The Committee fully endorses the value of the diastolic reading and would suggest repeated observations and greater rigidity of selection when the diastolic constantly exceeds 95 mm. Hg.

N.B. Your Committee recommends direction being given by the various companies to their Examiners, to read the diastolic pressure at the moment of disappearance of all sound.

This section was adopted with the request that a table of Diastolic Pressures be added.

MORTALITY RATINGS FOR BUILD AND AVERAGE FAMILY HISTORY

For some years the Canadian companies had been estimating extra mortality for risks which were sub-standard owing to overweight, by a method proposed by the Actuaries' Club of Toronto. This method was based on two principles: (1) That it was possible to obtain a mean weight irrespective of the age of the applicant and dependent only on the height, the extra mortality varying in proportion to the departure from this mean weight; (2) that the companies would allow an extra mortality of 20 per cent. without rating the risk.

In order to make this method adaptable to a numerical rating system, a table was constructed showing the mortality rating for weights above and below a mean weight. This table was found to agree very closely with the table adopted by one of the American companies, with the exception of the figures in the extreme overweight groups. It was, therefore, decided to adopt the exact figures as published by this American company, with the exception above mentioned of the extreme overweights.

MORTALITY RATINGS FOR BUILD AND AVERAGE FAMILY HISTORY

Height		Weight										Mean Weight	Weight										Height						
5-0	76	81	86	91	96	101	106	111	116	121	126	131	136	141	146	151	156	161	166	171	176	181	186	191	196	201	206	211	5-0
1	70	81	86	91	96	101	106	111	116	121	126	131	136	141	146	151	156	161	166	171	176	181	186	191	196	201	206	211	1
2	78	83	88	93	98	103	108	113	118	123	128	133	138	143	148	153	158	163	168	173	178	183	188	193	198	203	208	213	2
3	81	86	91	96	101	106	111	116	121	126	131	136	141	146	151	156	161	166	171	176	181	186	191	196	201	206	211	216	3
4	85	90	95	100	105	110	115	120	125	130	135	140	145	150	155	160	165	170	175	180	185	190	195	200	205	210	215	220	4
5	89	94	99	104	109	114	119	124	129	134	139	144	149	154	159	164	169	174	179	184	189	194	199	204	209	214	219	224	5
6	93	98	103	108	113	118	123	128	133	138	143	148	153	158	163	168	173	178	183	188	193	198	203	208	213	218	223	228	6
7	97	102	107	112	117	122	127	132	137	142	147	152	157	162	167	172	177	182	187	192	197	202	207	212	217	222	227	232	7
8	101	106	111	116	121	126	131	136	141	146	151	156	161	166	171	176	181	186	191	196	201	206	211	216	221	226	231	236	8
9	105	110	115	120	125	130	135	140	145	150	155	160	165	170	175	180	185	190	195	200	205	210	215	220	225	230	235	240	9
10	109	114	119	124	129	134	139	144	149	154	159	164	169	174	179	184	189	194	199	204	209	214	219	224	229	234	239	244	10
11	113	118	123	128	133	138	143	148	153	158	163	168	173	178	183	188	193	198	203	208	213	218	223	228	233	238	243	248	11
12	117	122	127	132	137	142	147	152	157	162	167	172	177	182	187	192	197	202	207	212	217	222	227	232	237	242	247	252	12
13	121	126	131	136	141	146	151	156	161	166	171	176	181	186	191	196	201	206	211	216	221	226	231	236	241	246	251	256	13
14	125	130	135	140	145	150	155	160	165	170	175	180	185	190	195	200	205	210	215	220	225	230	235	240	245	250	255	260	14
15	129	134	139	144	149	154	159	164	169	174	179	184	189	194	199	204	209	214	219	224	229	234	239	244	249	254	259	264	15
16	133	138	143	148	153	158	163	168	173	178	183	188	193	198	203	208	213	218	223	228	233	238	243	248	253	258	263	268	16
17	137	142	147	152	157	162	167	172	177	182	187	192	197	202	207	212	217	222	227	232	237	242	247	252	257	262	267	272	17
18	141	146	151	156	161	166	171	176	181	186	191	196	201	206	211	216	221	226	231	236	241	246	251	256	261	266	271	276	18
19	145	150	155	160	165	170	175	180	185	190	195	200	205	210	215	220	225	230	235	240	245	250	255	260	265	270	275	280	19
20	149	154	159	164	169	174	179	184	189	194	199	204	209	214	219	224	229	234	239	244	249	254	259	264	269	274	279	284	20
21	153	158	163	168	173	178	183	188	193	198	203	208	213	218	223	228	233	238	243	248	253	258	263	268	273	278	283	288	21
22	157	162	167	172	177	182	187	192	197	202	207	212	217	222	227	232	237	242	247	252	257	262	267	272	277	282	287	292	22
23	161	166	171	176	181	186	191	196	201	206	211	216	221	226	231	236	241	246	251	256	261	266	271	276	281	286	291	296	23
24	165	170	175	180	185	190	195	200	205	210	215	220	225	230	235	240	245	250	255	260	265	270	275	280	285	290	295	300	24
25	169	174	179	184	189	194	199	204	209	214	219	224	229	234	239	244	249	254	259	264	269	274	279	284	289	294	299	304	25
26	173	178	183	188	193	198	203	208	213	218	223	228	233	238	243	248	253	258	263	268	273	278	283	288	293	298	303	308	26
27	177	182	187	192	197	202	207	212	217	222	227	232	237	242	247	252	257	262	267	272	277	282	287	292	297	302	307	312	27
28	181	186	191	196	201	206	211	216	221	226	231	236	241	246	251	256	261	266	271	276	281	286	291	296	301	306	311	316	28
29	185	190	195	200	205	210	215	220	225	230	235	240	245	250	255	260	265	270	275	280	285	290	295	300	305	310	315	320	29
30	189	194	199	204	209	214	219	224	229	234	239	244	249	254	259	264	269	274	279	284	289	294	299	304	309	314	319	324	30
31	193	198	203	208	213	218	223	228	233	238	243	248	253	258	263	268	273	278	283	288	293	298	303	308	313	318	323	328	31
32	197	202	207	212	217	222	227	232	237	242	247	252	257	262	267	272	277	282	287	292	297	302	307	312	317	322	327	332	32
33	201	206	211	216	221	226	231	236	241	246	251	256	261	266	271	276	281	286	291	296	301	306	311	316	321	326	331	336	33
34	205	210	215	220	225	230	235	240	245	250	255	260	265	270	275	280	285	290	295	300	305	310	315	320	325	330	335	340	34
35	209	214	219	224	229	234	239	244	249	254	259	264	269	274	279	284	289	294	299	304	309	314	319	324	329	334	339	344	35
36	213	218	223	228	233	238	243	248	253	258	263	268	273	278	283	288	293	298	303	308	313	318	323	328	333	338	343	348	36
37	217	222	227	232	237	242	247	252	257	262	267	272	277	282	287	292	297	302	307	312	317	322	327	332	337	342	347	352	37
38	221	226	231	236	241	246	251	256	261	266	271	276	281	286	291	296	301	306	311	316	321	326	331	336	341	346	351	356	38
39	225	230	235	240	245	250	255	260	265	270	275	280	285	290	295	300	305	310	315	320	325	330	335	340	345	350	355	360	39
40	229	234	239	244	249	254	259	264	269	274	279	284	289	294	299	304	309	314	319	324	329	334	339	344	349	354	359	364	40
41	233	238	243	248	253	258	263	268	273	278	283	288	293	298	303	308	313	318	323	328	333	338	343	348	353	358	363	368	41
42	237	242	247	252	257	262	267	272	277	282	287	292	297	302	307	312	317	322	327	332	337	342	347	352	357	362	367	372	42
43	241	246	251	256	261	266	271	276	281	286	291	296	301	306	311	316	321	326	331	336	341	346	351	356	361	366	371	376	43
44	245	250	255	260	265	270	275	280	285	290	295	300	305	310	315	320	325	330	335	340	345	350	355	360	365	370	375	380	44
45	249	254	259	264	269	274	279	284	289	294	299	304	309	314	319	324	329	334	339	344	349	354	359	364	369	374	379	384	45
46	253	258	263	268	273	278	283	288	293	298	303	308	313	318	323	328	333	338	343	348	353	358	363	368	373	378	383	388	46
47	257	262	267	272	277	282	287	292	297	302	307	312	317	322	327	332	337	342	347	352	357	362	367	372	377	382	387	392	47
48	261	266	271	276	281	286	291	296	301	306	311	316	321	326	331	336	341	346	351	356	361	366	371	376	381	386	391	396	48
49	265	270	275	280	285	290	295	300	305	310	315	320	325	330	335	340	345	350	355	360	365	370	375	380	385	390	395	400	49
50	269	274	279	284	289	294	299	304	309	314	319	324	329	334	339	344	349	354	359	364	369	374	379	384	389	394	399	404	50
51	273	278	283	288																									

Age	Mortality Ratios (Average Family History)										Mortality Ratios (Average Family History)										Age
	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100		
15	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100		
20	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100		
25	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100		
30	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100		
35	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100		
40	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100		
45	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100		
50	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100		
55	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100		
60	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100		

DEAFNESS

Total—\$2 for Endowment, 20 years or less.

\$3 on all other plans.

Partial—From standard to above extras.

Deaf Mutes—\$3 for Endowment, 20 years or less.

\$5 for all other plans.

TOTAL DISABILITY TO BE DISALLOWED

The following are cases wherein total disability is to be disallowed:

- No. 1. Where the applicant has had a definite history of primary pleurisy. *Except* under conditions where a standard policy is granted and the case is not border-line.
- No. 2. Where the applicant has had a definite history of tuberculosis of any part of the body at any time in the past. (Lungs, bones, joints, glands, etc.)
- No. 3. Where the family history shows that there has been one or more deaths from tuberculosis among parents, brothers, or sisters. *Except* where a standard policy is granted and the case is not border-line.
- No. 4. Where the applicant has had a nervous breakdown.
- No. 5. Where there is a history of insanity in the direct family line.
- No. 6. Where the applicant has had an attack of acute articular rheumatism. *Except* under conditions where a standard policy is granted.
- No. 7. Where there has been loss of one eye, one foot, or one hand.
- No. 8. Where the applicant gives a history of syphilis.
- No. 9. Where the habits in regard to alcohol are doubtful.
- No. 10. Where the blood pressures are unduly increased.
- No. 11. Where the applicant engages in hazardous or unhealthful occupation. See schedule of occupations.
- No. 12. Where the environment renders applicant more than usually liable to total disability.

N. B. The Committee suggests that great care should be exercised in granting the Total Disability:

- (a) To young underweights.
- (b) In cases where standard policies are granted under Sections 1, 2, and 3, if applicant is under 30 years of age.
- (c) In case of illegitimates.

This section was approved with the suggestion that it might be necessary at a later date to prepare Disability rates to apply to certain types of sub-standard risks.

GOITRE

Exophthalmic—without operation:

At any time in the past or on examination—Decline.

Exophthalmic—with history of successful operation:

- (a) Within five years—Decline.
- (b) With freedom from all symptoms for five years after operation—Provide for Mortality of 135% at least.

Simple Goitre—without operation:

- (a) With any adverse symptoms upon examination—Decline.
- (b) With freedom from all symptoms for one year—120% and upwards.

Simple Goitre—with history of successful operation:

- (a) With any adverse symptoms upon examination, or within one year of the date of the operation—Decline.
- (b) With freedom from all symptoms for from one to five years after operation—125% and upwards.
- (c) With freedom from all symptoms for more than five years—Standard.

HEART MURMURS

Mitral Regurgitation, without Hypertrophy (no other impairment):

Life plans, accept at 165%.

ENDOWMENTS, 15 or 20 years, with slightly less mortality, suggest 150%.

Mitral Regurgitation, with Hypertrophy.

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Mortality Mean Ratio—205%. Cannot recommend safe basis for acceptance.

Mitral Regurgitation with History of Inflammatory Rheumatism—Decline.

Mitral Regurgitation with Irregular or Intermittent Pulse—Decline.

Functional heart murmurs, if carefully selected, are insurable among young applicants at standard rates; among applicants over 40 years of age, at rates calculated to provide for a substantial extra mortality. Suggest 140%.

This, it will be observed, is based on the Hunter-Rogers' investigation. Some of our Companies are dealing with the cases a little more freely and perhaps liberally than the schedule indicates, but only, or chiefly, where dependable fleuroscopic tracings may be had.

HERNIA

I. Inguinal and ventral, supported and unsupported no extra, except in cases which present some indication of extra danger.

II. Inguinal and double, supported and unsupported, mortality of 120%.

III. Femoral—mortality 125%.

IV. Irreducible hernia—150%.

EXCISION OF ONE KIDNEY

(A) When the operation has been undertaken for tuberculosis or malignant disease of the organ at any time in the past—Decline.

(B) For other affections—hydronephrosis, pyonephrosis, stone, etc.

(1) If application within two years of operation—defer until 2 years after operation.

(2) If application from 2 to 5 years of operation, and if remaining organ functioning perfectly, and if there have been no symptoms (pain, etc.) referable to it—provide for a mortality of 200%.

(3) If application after 5 years from operation, and if condition similar to above exists, provide for mortality of 175%.

5-0	5-1	5-2	5-3	5-4	5-5	5-6	5-7	5-8	5-9	6-0
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PLEURISY AND BUILD (COMBINED)

Height.....	5-0	5-1	5-2	5-3	5-4	5-5	5-6	5-7	5-8	5-9	5-10	5-11	6-0	6-1	6-2	6-3
Mean Weight.....	131	131	133	136	140	144	148	152	157	162	167	172	178	185	192	200

Years Elapsed Since Date of Attack																
Age	2	3	4	5	6	7	8	9	10	Years Elapsed Since Date of Attack						
	15	20	25	30	35	40	45	50	55	Years Elapsed Since Date of Attack						
-40 lbs.										Years Elapsed Since Date of Attack						
Age	15	20	25	30	35	40	45	50	55	Age	2	3	4	5	6	7
	15	20	25	30	35	40	45	50	55		2	3	4	5	6	7
-35 lbs.										Years Elapsed Since Date of Attack						
Age	15	20	25	30	35	40	45	50	55	Age	2	3	4	5	6	7
	15	20	25	30	35	40	45	50	55		2	3	4	5	6	7
-30 lbs.										Years Elapsed Since Date of Attack						
Age	15	20	25	30	35	40	45	50	55	Age	2	3	4	5	6	7
	15	20	25	30	35	40	45	50	55		2	3	4	5	6	7
-25 lbs.										Years Elapsed Since Date of Attack						
Age	15	20	25	30	35	40	45	50	55	Age	2	3	4	5	6	7
	15	20	25	30	35	40	45	50	55		2	3	4	5	6	7
-20 lbs.										Years Elapsed Since Date of Attack						
Age	15	20	25	30	35	40	45	50	55	Age	2	3	4	5	6	7
	15	20	25	30	35	40	45	50	55		2	3	4	5	6	7
-15 lbs.										Years Elapsed Since Date of Attack						
Age	15	20	25	30	35	40	45	50	55	Age	2	3	4	5	6	7
	15	20	25	30	35	40	45	50	55		2	3	4	5	6	7
-10 lbs.										Years Elapsed Since Date of Attack						
Age	15	20	25	30	35	40	45	50	55	Age	2	3	4	5	6	7
	15	20	25	30	35	40	45	50	55		2	3	4	5	6	7
-5 lbs.										Years Elapsed Since Date of Attack						
Age	15	20	25	30	35	40	45	50	55	Age	2	3	4	5	6	7
	15	20	25	30	35	40	45	50	55		2	3	4	5	6	7
0 lbs. to +5 lbs.										Years Elapsed Since Date of Attack						
Age	15	20	25	30	35	40	45	50	55	Age	2	3	4	5	6	7
	15	20	25	30	35	40	45	50	55		2	3	4	5	6	7
+10 lbs. to +25 lbs.										Years Elapsed Since Date of Attack						
Age	15	20	25	30	35	40	45	50	55	Age	2	3	4	5	6	7
	15	20	25	30	35	40	45	50	55		2	3	4	5	6	7
+30 lbs. or more										Years Elapsed Since Date of Attack						
Age	15	20	25	30	35	40	45	50	55	Age	2	3	4	5	6	7
	15	20	25	30	35	40	45	50	55		2	3	4	5	6	7

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This table was criticised on the grounds;—

(1) That ten years was too long a period from the date of the attack to impose a rating.

(2) That the rating was too heavy for those of approximate average weight.

Other criticism of this table was made, but it was decided to adopt it for a year's trial, it being understood that the companies should not be asked to follow it if it did not meet with their approval.

RENAL COLIC, DEFINITE HISTORY OF, WITH OR WITHOUT OPERATION

With operation for removal of stone, or a single attack of renal colic, without operation, and despite history of having passed stone or gravel:

If application within 1 year.... Preferable to defer for at least a year from date of attack or operation, but if taken to provide for at least 150% mortality.

If application from 1 to 2 years after date of attack or operation..... Provide for 135% mortality.

If application from 2 to 5 years after date of attack or operation..... Provide for 120% mortality.

If application more than 5 years after attack or operation..... Standard.

Repeated attacks without operation..... Committee cannot recommend a rating.

N.B. Attention is directed to a markedly increased hazard not met by suggested ratings when the applicant is overweight.

RHEUMATISM (Definite History of Acute Articular)

Group (1)

One Attack

Postpone until at least one year has elapsed since recovery from attack.

From 1-5 years—Mortality at least 125%.

Over 5 years—Standard.

Group (2)

Two Attacks

(a) Postpone for two years from date of recovery from last attack.

(b) Last from 2-5 years ago, at least 135%.

Last from 5-10 years, 125%.

Last over 10 years ago—Standard.

More than Two Attacks.—Cannot recommend rating.

SUGAR

The following procedure is recommended:

1. When found by Medical Examiner, remainder of specimen, preserved with boric acid, should be forwarded to Head Office whenever possible. If verified, and also in cases where there is an insurance history of sugar, ask for another specimen, taken between one and two hours after heaviest meal.
2. If found again, postpone one year.
3. If not found and the applicant is still willing to proceed on the understanding that he may not receive a standard policy, a Sugar Tolerance Test is to be given, preferably glucose, 50 grams, after a meal (otherwise two consecutive heavy starch and sugar meals), the specimens to be collected one and four hours after the meal and glucose, or after the second "starch and sugar" meal. Preserve the specimens with boric acid and forward to Head Office.
4. At the end of probationary period, if the postponed applicant is willing to proceed on the understanding that he may not receive standard insurance, and if a sample of

urine is now free of sugar, a Sugar Tolerance Test is to be given as indicated under paragraph 3.

5. Blood Sugar Tests should be had wherever possible and reliable.
6. The examiner should be instructed to catechise the applicant as to whether he is now or has been on restricted diet.
 - (a) Rigid selection—older cases more favorable, but watch for arteriosclerosis.
 - (b) Provision for extra mortality in all but most favorable cases.
 - (c) Desirable to limit risks to small amounts of insurance.
7. If applicant *dieting* reject.

PRACTICE

1. History of one attack over five years—disregarded, if three ordinary tests (one of a specimen taken after the heaviest meal in the day) are clear.
2. Sugar found once on examination or history of one attack within five years—submit to Tolerance Test.
 - (a) If clear—120% up.
 - (b) If trace of sugar found in one-hour sample, free in four-hour sample, and if Blood Sugar Test satisfactory—125% up. If Blood Sugar Test not obtained—150% up.
 - (c) If sugar found in one and four-hour samples—R. N. A.
3. History of two attacks within five years, last prior to one year—submit to Tolerance Test.
 - (a) If clear—135% up.
 - (b) If trace of sugar found in one hour sample and free in four-hour sample, and if the Blood Sugar Test satisfactory—150% up. If Blood Sugar Test not obtained—cannot recommend rating.
 - (c) If sugar in both samples—R. N. A.

Scadding—Impaired Risks

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TUBERCULOSIS IN FAMILY HISTORY

BASIC RATINGS FOR ONE CASE

Departures from mean weight:

Age	45	40	35	30	25	20	15	10	5	0	PLUS	5	10	15	20	25
15	75	70	65	60	55	50	45	40	35	30		25	20	15	10	5
20	65	60	55	50	45	40	35	30	25	20		15	10	10	5	0
25	50	45	40	40	35	30	25	20	15	15		10	5	0		
30	40	35	30	25	20	20	15	15	10	10		5	0			
35	35	30	25	20	15	15	10	10	5	5						
40	25	20	15	10	10	5	5	0	0	0						
45	20	15	10	10	5	5	0	0	0	0						
50	15	10	10	5	5	0	0	0	0	0						

TUBERCULOSIS IN FAMILY HISTORY

BASIC RATINGS FOR TWO CASES

Departures from mean weight:

Age	45	40	35	30	25	20	15	10	5	0	PLUS	5	10	15	20	25	30	35
15	100	90	80	75	70	65	60	55	50	45		40	35	30	20	15	10	5
20	85	75	65	60	55	50	45	40	35	30		25	20	15	10	5	0	
25	65	60	55	50	45	40	35	30	25	20		15	10	10	5	0	0	
30	55	50	45	40	35	30	25	20	15	15		10	10	5	0			
35	50	45	40	35	30	25	20	15	10	5		5	0					
40	40	35	30	25	20	15	10	5	0	0								
45	25	20	15	15	10	10	5	5	0	0								
50	20	15	10	10	5	5	0	0	0	0								

There was considerable criticism of this table, particularly with reference to the ratings imposed for a medium departure from average weight, and for ages over thirty. After a great deal of discussion, the table was referred to the Committee for reconsideration, power being given to the Committee to add to its numbers.

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ULCER, GASTRIC AND DUODENAL

Gastric.—Postpone three years from the date of full recovery from attack or operation for relief.

From 3 to 5 yrs.	175%	mortality
" 5 to 7 "	150%	"
" 7 to 10 "	125%	"
After 10 years	120%	"

The Committee regards such ratings in the whole class, whether operated upon or not, as experimental, and suggests that the relative amount of insurance be much more severely limited than is the usual practice in sub-standard cases.

Duodenal.—Refer at least two years from the date of full recovery from attack or operation for relief.

From 2 to 5 yrs. provide for mortality of	150%
" 5 to 7 " " " " "	125%
After 7 years—standard—mortality	115%

The Committee regards such ratings in the whole class, whether operated upon or not, as experimental, and suggests that the relative amount of insurance be much more severely limited than is the usual practice in sub-standard cases.

The suggestion was made by one of the members that the probationary period be two years instead of three years, but this motion was lost and the section as submitted was approved.

**ADVANCES IN AGE TO MEET THE EXTRA HAZARD ON SUB-
STANDARD LIVES FOR VARIOUS PERCENTAGES
OF EXTRA MORTALITY**

This table is based on the Om. (5) Table of Mortality with interest at three and one-half per cent.

10, 15 AND 20-YEAR ENDOWMENTS

Mortality Rating

Age at Entry	125	130	135	140	145	150	162½	175	200	250
15	13	14	15	17	18	19	21	23	26	30
6	13	14	15	16	17	18	20	23	25	29
7	12	13	14	16	17	18	19	21	24	28
8	11	12	13	15	16	17	18	20	23	27
9	11	12	13	14	15	16	18	20	23	27
20	10	11	12	13	14	15	17	19	22	26
1	10	11	12	13	14	15	17	18	21	25
2	9	10	11	12	13	14	16	18	20	24
3	9	10	11	12	13	14	16	17	20	24
4	8	9	10	11	12	13	16	17	19	23
25	8	9	10	11	12	13	15	16	19	22
6	8	9	10	10	11	12	14	15	18	22
7	7	8	9	10	11	12	14	15	18	21
8	7	8	9	9	10	11	13	14	17	21
9	7	8	9	9	10	11	13	14	16	20
30	6	7	8	8	9	10	12	13	16	19
1	6	7	8	8	9	10	12	13	15	19
2	6	7	8	8	9	10	11	12	15	18
3	6	7	7	8	8	9	11	12	14	18
4	5	6	7	7	8	9	10	12	14	17
35	5	6	7	7	8	9	10	11	13	17
6	5	6	6	7	7	8	10	11	13	16
7	5	6	6	7	7	8	10	11	13	16
8	5	6	6	7	7	8	9	11	12	16
9	4	5	6	6	7	8	9	11	12	15
40	4	5	5	6	6	7	9	10	12	15
1	4	5	5	6	6	7	8	10	12	15
2	4	5	5	6	6	7	8	9	11	14
3	4	5	5	6	6	7	8	9	11	14
4	4	5	5	6	6	7	8	9	11	14
45	4	4	5	5	6	6	8	9	11	14
6	4	4	5	5	6	6	7	9	10	13
7	3	4	4	5	5	6	7	8	10	13
8	3	4	4	5	5	6	7	8	10	13
9	3	4	4	5	5	6	7	8	10	13
50	3	4	4	5	5	6	7	8	10	13
1	3	4	4	5	5	6	7	8	10	12
2	3	4	4	5	5	6	7	8	9	12
3	3	4	4	5	5	6	7	8	9	12
4	3	4	4	5	5	6	7	8	9	12
55	3	3	4	4	5	5	7	8	9	12
60	3	3	4	4	5	5	7	8	9	12

ADVANCES IN AGE TO MEET THE EXTRA HAZARD ON SUB-
STANDARD LIVES FOR VARIOUS PERCENTAGES OF
EXTRA MORTALITY

*This table is based on the Om. (5) Table of Mortality with interest at
three and one-half per cent.*

25-YEAR ENDOWMENTS AND OVER

Mortality Ratings

Age at Entry	125	130	135	140	145	150	162½	175	200	250
15	11	12	13	14	15	16	18	20	23	27
6	10	11	12	13	14	15	17	19	22	26
7	10	11	12	13	14	15	17	19	21	25
8	9	10	11	12	13	14	16	18	21	25
9	9	10	11	12	13	14	16	18	20	24
20	8	9	10	11	12	13	15	17	19	23
1	8	9	10	11	12	13	15	16	19	23
2	8	9	10	10	11	12	14	16	18	22
3	7	8	9	10	11	12	14	15	18	21
4	7	8	9	9	10	11	13	15	17	21
25	7	8	9	9	10	11	13	14	16	20
6	6	7	8	9	10	11	13	14	16	20
7	6	7	8	9	10	11	12	13	15	19
8	6	7	8	8	9	10	12	13	15	19
9	6	7	8	8	9	10	11	12	15	18
30	6	7	7½	8½	8	9	11	12	14	18
1	5	6	7½	7½	8	9	11	12	14	17
2	5	6	6	7	7	8	10	11	13	17
3	5	6	6	7	7	8	10	11	13	16
4	5	6	6	7	7	8	10	11	13	16
35	4	5	6	6	7	8	9	10	12	16
6	4	5	5	6	6	7	9	10	12	15
7	4	5	5	6	6	7	9	10	12	15
8	4	5	5	6	6	7	9	10	12	15
9	4	5	5	6	6	7	8	9	11	15
40	4	5	5	6	6	7	8	9	11	14
1	4	5	5	6	6	7	8	9	11	14
2	4	4	5	5	6	6	8	9	11	14
3	4	4	5	5	6	6	8	9	10	13
4	3	4	4	5	5	6	7	8	10	13
45	3	4	4	5	5	6	7	8	10	12
6	3	4	4	5	5	6	7	8	10	12
7	3	4	4	5	5	6	7	8	10	12
8	3	4	4	5	5	6	7	8	10	12
9	3	4	4	5	5	6	7	8	10	12
50	3	3	4	4	5	5	7	8	10	12
55	3	3	4	4	5	5	6	7	9	11
60	3	3	4	4	5	5	6	7	9	11

**ADVANCES IN AGE TO MEET THE EXTRA HAZARD ON SUB-
STANDARD LIVES FOR VARIOUS PERCENTAGES OF
EXTRA MORTALITY**

This table is based on the Om. (5) Table of Mortality with interest at three and one-half per cent.

WHOLE LIFE WITH PREMIUMS LIMITED TO 10, 15 AND 20 YEARS

Mortality Rating

Age at Entry	125	130	135	140	145	150	162½	175	200	250
15	7	8	9	10	11	12	14	16	19	24
6	7	8	9	9	10	11	13	15	18	23
7	6	7	8	9	10	11	13	15	18	23
8	6	7	8	9	10	11	13	15	18	22
9	6	7	8	8	9	10	12	14	17	22
20	6	7	8	8	9	10	12	14	17	21
1	6	7	8	8	9	10	11	13	16	21
2	5	6	7	8	9	10	11	13	16	21
3	5	6	7	8	9	10	11	13	16	20
4	5	6	7	7	8	9	11	13	15	20
25	5	6	7	7	8	9	10	12	15	19
6	5	6	7	7	8	9	10	12	15	19
7	5	6	7	7	8	9	10	12	14	18
8	5	6	6	7	7	8	10	12	14	18
9	5	6	6	7	7	8	9	11	14	18
30	4	5	6	7	7	8	9	11	13	17
1	4	5	6	6	7	8	9	11	13	17
2	4	5	6	6	7	8	9	11	13	17
3	4	5	6	6	7	8	9	10	13	16
4	4	5	5	6	6	7	9	10	12	16
35	4	5	5	6	6	7	9	10	12	16
6	4	5	5	6	6	7	9	10	12	15
7	4	5	5	6	6	7	8	10	12	15
8	4	5	5	6	6	7	8	9	12	15
9	4	5	5	6	6	7	8	9	11	15
40	4	5	5	6	6	7	8	9	11	14
1	4	5	5	6	6	7	8	9	11	14
2	4	4	5	5	6	6	8	9	11	14
3	4	4	5	5	6	6	7	9	11	14
4	4	4	5	5	6	6	7	8	11	13
45	3	4	4	5	5	6	7	8	10	13
6	3	4	4	5	5	6	7	8	10	13
7	3	4	4	5	5	6	7	8	10	13
8	3	4	4	5	5	6	7	8	10	13
9	3	4	4	5	5	6	7	8	10	13
50	3	4	4	5	5	6	7	8	10	13
1	3	4	4	5	5	6	7	8	10	12
2	3	3	4	4	5	6	7	8	10	12
3	3	3	4	4	5	5	6	8	10	12
4	3	3	4	4	5	5	6	7	10	12
55	3	3	4	4	5	5	6	7	10	12
60	3	3	4	4	5	5	6	7	10	12

ADVANCES IN AGE TO MEET THE EXTRA HAZARD ON SUB-
STANDARD LIVES FOR VARIOUS PERCENTAGES OF
EXTRA MORTALITY

*This table is based on the Om. (5) Table of Mortality with interest at
three and one-half per cent.*

WHOLE LIFE WITH PREMIUMS PAYABLE THROUGHOUT LIFE OR LIMITED
TO 25 YEARS AND OVER

Mortality Rating

Age at Entry	125	130	135	140	145	150	162½	175	200	250
15	6	7	8	9	10	11	13	14	17	22
6	6	7	8	9	10	11	13	14	17	22
7	6	7	8	8	9	10	12	14	17	21
8	5	6	7	8	9	10	12	13	16	21
9	5	6	7	8	9	10	11	13	16	20
20	5	6	7	8	9	10	11	13	16	20
1	5	6	7	7	8	9	11	13	15	20
2	5	6	7	7	8	9	10	12	15	19
3	5	6	7	7	8	9	10	12	15	19
4	5	6	7	7	8	9	10	12	14	18
25	5	6	7	7	8	9	10	11	14	18
6	5	6	6	7	7	8	10	11	14	18
7	5	6	6	7	7	8	10	11	13	17
8	4	5	6	6	7	8	10	11	13	17
9	4	5	6	6	7	8	10	11	13	17
30	4	5	6	6	7	8	9	10	13	16
1	4	5	5	6	6	7	9	10	12	16
2	4	5	5	6	6	7	9	10	12	16
3	4	5	5	6	6	7	9	10	12	16
4	4	5	5	6	6	7	9	10	12	15
35	4	5	5	6	6	7	8	9	12	15
6	4	5	5	6	6	7	8	9	11	15
7	4	5	5	6	6	7	8	9	11	15
8	4	5	5	6	6	7	8	9	11	14
9	4	4	5	5	6	6	8	9	11	14
40	4	4	5	5	6	6	8	9	11	14
1	3	4	4	5	5	6	8	9	11	14
2	3	4	4	5	5	6	7	8	10	14
3	3	4	4	5	5	6	7	8	10	13
4	3	4	4	5	5	6	7	8	10	13
45	3	4	4	5	5	6	7	8	10	13
6	3	4	4	5	5	6	7	8	10	13
7	3	4	4	5	5	6	7	8	10	13
8	3	4	4	5	5	6	7	8	10	13
9	3	4	4	5	5	6	7	8	10	13
50	3	4	4	5	5	6	7	8	10	12
1	3	4	4	5	5	6	7	8	9	12
2	3	4	4	5	5	6	7	8	9	12
3	3	3	4	4	5	5	7	8	9	12
4	3	3	4	4	5	5	6	7	9	12
55	3	3	4	4	5	5	6	7	9	12
60	3	3	4	4	5	5	6	7	9	12

As indicated before, it is quite understood by all the companies that the whole schedule is to be regarded as a guide and not an inflexible rule of set standards.

Further discussions and suggestions for more satisfactory treatment are invited at any Annual Meeting, not only regarding impairments which have been dealt with solely by the Committee, but also as to the impairments which have been recommended at any Annual Meeting of the whole body of representatives. The trial of any method of treatment for the period of a year by a number of companies should enable us to obtain a better collective idea as to the practicability and fairness of such method.

Dr. McMahon—Dr. Hamilton will lead the discussion of this paper.

Dr. Hamilton—Mr. President and Gentlemen: I have been able on but one occasion to sit with the gentlemen who have been discussing these very important questions referred to in Dr. Scadding's paper.

The report just received represents a very tedious bit of work, yet withal a work that has been carried on with great enthusiasm and good judgment at the sessions in Toronto. Dr. Scadding's paper has stimulated us all along various lines of investigation, but this investigation must go on much further if results are to be reached, even though the sum total of our companies in Canada is small when compared with those in the larger companies of this country.

A particular group of cases which practically all companies must consider and which our actuaries in the Sun Life have recently studied, are those with history of pleurisy. Apparently all these are cases of impairment, and in doing substandard work we have tried to anticipate this impairment as closely as possible. It would appear, however, according to the figures which Mr. Mabon, our Assistant Actuary, has supplied, that we have failed to meet the actual mortality rate by our rating. Taking a group of upwards of 200, covering a

period of about fifteen years, it has been found that the mortality rate has exceeded that anticipated to a considerable degree—the group showing a mortality of 174%. Many of the cases have died of tuberculosis in the first five or seven years. By this study we are thus compelled to give more careful selection to cases showing such impairment. There seems to be no doubt whatever that a numerical expression of impairments is the best guide to a decision, and Medical Referees owe much to the painstaking work of our actuaries who are doing so much to point the way and render assistance in all bewildering problems.

I wish to add that our blood pressure experiences are being compiled but at present we can give no report upon them. We have all been greatly helped by the expression of opinion on this subject which this meeting has afforded.

Dr. Thos. D. Archibald: When our President asked me to discuss this paper I felt obliged to reply that it was a poor sheep dog that worried his own sheep, as I had the honor and pleasure of acting on Dr. Scadding's Committee. At the outset I should like to pay tribute before this meeting to Dr. Scadding for his zeal, untiring energy, and patience in carrying out the work, the result of which you have heard. He may very justly be called the "Rogers" of Toronto.

The principle of Companies getting together in order to reach some more or less uniform basis on which to deal with substandard risks, must appeal to all of us. If the idea is practical—and at the present time most of the Canadian Companies are following the committee's suggestions—the results so far as the actual to expected mortality are concerned should in a few years be of very material value. These results will enable us to find out in a much shorter time mistakes which we may now be making in dealing too leniently with one group, or too severely with another.

I do not feel qualified in any way to pass opinion upon any of the various groups individually. In fact, after hearing in committee each subject hashed and re-hashed I have yet a distinct sensation of mental indigestion and can again only

return thanks to our Chairman, Dr. Scadding, for finally insisting that the discussion be closed and some conclusion arrived at, which meant that our worthy colleagues, the actuaries, were asked to prepare a mortality table.

Amputations:

So far as amputations are concerned it must frankly be admitted that aside from the extra accident hazard our rating is purely a guess. We have had quite a large number of applicants in this group since the war and while they are probably living under different circumstances than the ordinary pre-war accident amputation case, we feel that an extra mortality is bound to be experienced.

Asthma:

It is, of course, understood that in applying the suggested ratings in this group that the greatest precaution must be taken to exclude asthmas which might be merely symptoms of cardiac or renal disease.

Goitre:

It is possible that this particular group has been in some instances too harshly dealt with. Undoubtedly, there are cases of exophthalmic goitre, which have not undergone operation but where by medicinal or X-ray treatment the symptoms have been negative for a long period of time, which are safely insurable with a proper loading.

Pleurisy:

Aside from the albumen and sugar groups there is no class which, perhaps, causes more difficulty than applicants who have had pleurisy. The chief trouble arises in establishing the fact as to whether the condition was a frank primary pleurisy. So many cases of pleurodynia are wrongly called pleurisy. Also, since the investigations, so thoroughly carried out during war service, our opinion has changed very materially on the question of tuberculosis being the chief factor in pleurisy with

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effusion. The whole question really is whether or not the condition was of tubercular origin. As this is impossible to determine in practically ninety per cent of the cases, we really felt that it was absolutely necessary to "play safe." You will notice in the table that the underweights at the younger ages are very heavily loaded.

Rheumatism:

While we will probably all agree in general with the ratings imposed—although from a perusal of the paper by Dr. Rogers and Mr. Hunter I must confess that we seem to have been far too lenient—I feel personally, that where there has been a definite focal infection which has been eradicated that certain cases with other especially favorably features can be raised out of the group. This is particularly true in cases where the applicant has suffered from two or more attacks, the cause of which is then discovered and removed, and followed by a reasonable time of freedom from further trouble.

Dr. Patton—Mr. President and Gentlemen: I was very glad to be present at the meeting last June in Toronto, and I am very much interested in the work they have been doing. I felt that they had made distinct progress and had reached a stage whereby they might begin to place their information in line with the information obtained by the American companies, recognizing that certain climatic and living conditions there differed from a large portion of the States, and that would make some difference in some of their experience. Undoubtedly already they have been acting along the lines in which their medical men and actuaries have been working together, which is absolutely essential in any business. I was very glad to see, too, that the lines on which we had been handling our Canadian business were very close to the lines worked out by them from their own experience.

I was impressed with the earnestness and the ability with which they were handling the work and I was very glad indeed to have had the opportunity to be with them.

Dr. Archibald has referred to the assistance we may expect

from Insulin. The work of Banting and his associates is epoch-making. Enormous difficulties have been encountered and surmounted and it is hoped much more will be accomplished in behalf of the diabetic. I very much fear that the discovery will not give us, as medical advisers, *immediate* relief in dealing with one of the most difficult impairments, inasmuch as it would appear that so far tolerance has not been permanently influenced by Insulin.

It is fortunate that the proportion of risks in which sugar is found is small. The concern such a finding gives is great. Surely there is no single impairment to compare with this in convincing any medical adviser that his own necessarily limited experience is of little worth. The experience of one medical practitioner may have been favorable and happy, another's unfavorable and appalling.

We hear it said that this or that risk is not a diabetic or potential diabetic and insurable at tabular rates because, though the applicant shows increase in blood sugar and marked glycosuria following a glucose meal, he deals quickly with that; the fact merely indicating a highly permeable kidney filter. If this be true, the question arises—will individuals possessing organs of this type live as long as those who can carry an occasional overload of blood sugar without spilling it?

Unless it can be established that every normal individual at some time may exhibit sugar in the urine, a single definite finding is the evidence of a defective chemistry due to a pancreas unequal at times to its task. Are we not in such a case dealing with an elementary glycosuria, in which the rate of progress is problematical to a degree?

Dr. Dwight has shown very clearly the result of a liberal selection in his own company and what a rigid selection, with its "painful irritation of the agency forces," can accomplish.

Accurate blood and urine tests facilitate the forecast of urban cases, usually involving the larger risks, but what we are most in need of is a measuring rod by which to select those cases coming from districts served by examiners incapable of expert blood analyses. It may be actuarially correct but, in a

country having a farmers' government, it is as much as a company's life is worth, if such government knew it, to penalize more severely a rural applicant who cannot be minutely examined in order to even up on his over-fed, possibly artful, but better examined brother!

I was very interested in Dr. Hamilton's remarks about pleurisy and only regret he was not able to present his company's findings at our last Annual Meeting. I think there were some actuaries and a few business men and perhaps some medical advisers present at that meeting who were rather inclined to think that our Pleurisy and Build Table was a little too stiff. In the light of this experience of Dr. Hamilton's company, I dare say we shall all feel more content to follow the guide as it stands for a little while longer.

Dr. McMahon—Dr. Exton will present to us a Simple and Rapid Test for Albumin, but I understand he has first something to say about a test for sugar.

PRELIMINARY COMMUNICATION OF A METHOD
FOR ESTIMATING THE SIGNIFICANCE
OF GLYCOSURIA

BY WILLIAM G. EXTON, M.D.,

Director Prudential Laboratory

During the course of a year we receive more inquiries regarding the significance of sugar in the urine than any other problem with which we have to deal, and during the latter part of this year I have reluctantly been obliged to write letters explaining that we were working on something which we were not at liberty to communicate.

I am therefore happy to be able to tell you that the Harri-man Research Laboratory in New York City has given me its

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consent to make a sort of preliminary communication, as it were, at the present time, of a method which looks to be very promising. In fact it appears very probable that we may be able to use it in life insurance work as a substitute for the blood sugar, and that possibly when it has been more thoroughly worked out in the light of investigations which are now being made of other phases of carbohydrate intolerance, the method may be developed into an accurate means of gauging the significance of sugar in the urine in a given case so as to enable one to decide on reasonable grounds as to whether a particular individual showing sugar in the urine is diabetic or not.

I would like to have it perfectly understood that Dr. Sharlit of the Harriman Research Laboratory developed the idea and has done most of the work in relation to this method, and that we have been carrying on an investigation with him using several different types of individuals, *i. e.*, insurance applicants, policyholders who use our longevity service, and some skillful diabetic patients showing sugar-free urines through the kindness of a prominent specialist, who is interested in the insurance as well as every other slant on diabetes.

There is a very impressive and complicated background of experimental work which I will try to avoid as much as possible in order to tell you as simply as I can the rationale of the procedure.

Let us begin with the Benedict's qualitative test for sugar in urine. In this we have a very satisfactory test for sugar, and in saying sugar we must always remind ourselves that this does not specifically mean glucose. When we talk of sugar we mean all of the saccharides which reduce copper, because for years it has been customary to speak of such clinically as sugar. With Benedict's qualitative test then we are able to measure down to two tenths of 1% of sugar and that is definitely the end point of sensitiveness of Benedict's qualitative test.

During the last year or two several tests have been developed which measure what might be called the subliminal or the so-

called normal urinary sugar, and these tests will show sugar in the urine when the concentration is less than two tenths of 1%. The best known tests of this type are Folin and Berglund's, Schaeffer and Hartman's, and Benedict's test for normal sugar in urine, and the latter will be found somewhat more handy to make than the others.

When he published this test Professor Benedict stated that normal individuals excrete less than $1\frac{1}{2}$ grams of sugar during 24 hours, and he predicted at that time that $1\frac{1}{2}$ grams might be taken as a very high normal maximum. Later experimental work has proven that Professor Benedict's prediction was correct. Now, a diabetic, such a one who will try to get by us by dieting himself, is able very easily to arrange his output of sugar so that he can hide his carbohydrate intolerance by excreting his sugar conveniently diluted so that it will not show by Benedict's, Haines' or Fehling's qualitative tests. The subliminal tests, however, enable us to measure the sugar in his urine, even when greatly diluted.

A vast amount of experimental work has been done on the sugar of blood and of normal urine as estimated by these so-called subliminal tests and all of the workers agree that all normal individuals show some degree of what Benedict has called "glycuresis" after an ordinary mixed meal. In other words we, all of us non-diabetic ones show a sugar excretion curve after meals which goes up rather sharply and comes down rather abruptly. Now diabetics who eat the same meal will show curves that go up higher and which fall more slowly. That is why Allen has given post-prandial hyperglycemia as the earliest practicable sign of diabetes.

This is a matter of practical importance for us to remember and worth while pointing out because many of us who use test meals do not always appreciate how important it is to get the urine at a particular time after the test meal. You may be able to spot a diabetic with a specimen of urine voided about **two** hours after a test meal when he will be able to get by you easily on a specimen passed several hours later. Test meals therefore will give safer results when the examiner arranges to

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get a specimen voided between $1\frac{1}{2}$ and $2\frac{1}{2}$ hours after the applicant has taken his test meal.

Another important point is to make certain that the applicant voids all of the urine in his bladder before he eats the test meal so that the specimen we get represents his renal secretion following the meal and not an admixture of starvation excretion.

Now, the sugar excretion curves characteristic of Benedict's or one of the other subliminal tests being definitely known both for normal individuals, and for diabetics, Dr. Sharlit glimpsed that if you could find some other element to measure in the urine it would throw a light on the concentration of the specimen you were dealing with and enable you to distinguish in a great many instances whether you were dealing with a diabetic mechanism or not; also that this light given by another element in the same specimen as we were testing for sugar, would be particularly useful in instances such as those with which we have to deal in which judgment must be based on random or chance specimens.

Now nitrogen is an element which may be used as such a light. Nitrogen is eliminated much more slowly than sugar. When proteins are eaten the nitrogen elimination rises to its peak at about four hours instead of two hours, as does the sugar. Therefore, if you estimate the total nitrogen which is not at all a difficult matter, and the subliminal sugar of the same sample of urine you have in the relationship of the sugar to the nitrogen something upon which you can predicate either how far removed from the intake of food the sample is representative, or the degree of concentration of the specimen. For instance, if you get a sample which shows a great deal of nitrogen and the same sample shows only a faint trace of sugar, in other words a low ratio, you will know that the man who passed the sample is not trying to fool you and that he is eating rather heavily, and that you are not dealing with a diabetic mechanism, because if he were diabetic and ate heavily enough to show such a high nitrogen content he should show a good deal more sugar in his urine.

There are many theoretical considerations involved in the relationship between the total nitrogen and the normal sugar of urine which I will not go into now, but it may be said that if you take the maximum normal sugar output, as given by Benedict which is $1\frac{1}{2}$ grams in 24 hours, and contrast this with the lowest nitrogen output that it is possible for any person to excrete on any kind of diet, which is about 6 grams in 24 hours, you get a theoretical ratio, and all samples of urine giving a sugar nitrogen ratio higher than this theoretical ratio would certainly be indicative of a diabetic condition. There is another ratio which has been arrived at by very interesting investigations and dietary experiments made by Dr. Sharlit and Dr. Lyle of the Harriman Research Laboratory, and some of their results are as follows:

In three young men under 30 years of age, living on an unrestricted mixed diet, the urine samples were taken for one week, two a day from each person. All of these samples represented urine excreted two hours after meals. In these normal individuals the ratio of sugar to nitrogen in no specimen exceeded 9%.

A number of urine samples passed two to four hours after carbohydrate test meals taken by normal individuals showed in no specimen a sugar to nitrogen ratio exceeding 10%.

A series of 24-hour samples of urine were had from a normal individual who lived part of the time on an extremely high protein diet, who then switched to starvation and abruptly followed starvation with a very high carbohydrate diet. Of the many samples which he passed on these different and extreme diets none of them showed a sugar to nitrogen ratio above 10%.

At the present time I feel that we have in this method of determining the ratio of normal sugar to total nitrogen a very promising test and one that may prove as sensitive as the blood sugar. Certainly the amount of nitrogen in the same specimen that shows traces of sugar is very informative.

I am not able at this time, nor as a matter of fact, has it yet been absolutely determined what the basic ratios are which

indicate intolerance to carbohydrates or diabetes. These must be worked out by further experiments, and I regret that I have not yet had even the time to tabulate our own cards because our own material shows some very interesting results.

This is merely, as stated in the beginning, a preliminary communication. We expect to go on and study the method and work out whatever values it may have for detecting and grading pre-diabetics and diabetics and communicate the results to you later.

A SIMPLE AND RAPID TEST FOR ALBUMIN IN URINE

BY WILLIAM G. EXTON, M. D.

Director, Prudential Laboratory

Last year I had the privilege of acquainting you with some of the details of our albumin work and it may be recalled that for some years we have been trying to develop a method of measuring the amounts of albumin in urine which would be accurate and rapid enough for routine work.

It seemed to us that such a test would not only help to eliminate a great deal of confusion and misinterpretation but that it would also form the basis for mathematically expressing the quantity of albumin in correlation with the concentration of the specimen. The degree of dilution of the specimen being a function of its albumin content a mathematical expression correlating these would afford a number of advantages and eventually tell us definitely what quantities of albumin might safely be regarded as significant or insignificant for each point of the specific gravity scale. Such an expression would also enable us to make more precise distinctions between cases of persistent albuminuria and cases which are now very loosely and, I fear, often mistakenly called intermittent albuminuria. From a Life Insurance standpoint there is nothing of greater importance than this in the whole consideration of albuminuria, because while it is true, as I have pointed

out on occasions, that albuminuria does not necessarily mean nephritis the hard, cold, inescapable fact remains that albuminuria is almost always the earliest sign of nephritis and is generally discoverable months and years before the most sensitive of renal function tests, or even the blood chemistry become diagnostic.

As a matter of technical practice we are accustomed to the use of criteria which do not truthfully inform us regarding the accidental, intermittent or persistent character of an albuminuria. For instance, if one uses Heller's test as the criterion, as most of us do, one is likely to miss altogether or to appraise as negligible the fainter reactions found in specimens of lower gravity, that is, gravities under about 1016, when the same amount of albumin in proportion to the concentration of the specimen would have appeared distinctly unfavorable if the gravity of the specimen happened to be 1028. Observation of nephritics or a few dilution experiments will demonstrate this point to the satisfaction of anyone and teach that in our daily work we are employing standards which cause us to classify as intermittent albuminurias cases which are as a matter of fact persistent albuminurias and only apparently intermittent.

How illusory and gross are the quantitative deductions we have been drawing from the reactions exhibited by the more commonly used albumin tests, a few very simple experiments, if carefully made, will show and it was therefore thought that a closer and more truthful means of dealing with albuminuria would prove of decided value.

Much has been written about what Cammidge has aptly called the "pitfalls" of Heller's and the heat test, and it is well known that the text books teach us not to rely on any one albumin test but to check up with one or two other tests. Thus, Heller's and the heat test in some one of its modifications have come to be preferred and to be employed far more frequently than other tests, although strangely enough they have in common some of the same "pitfalls."

Both of these tests serve their purpose beautifully if used

understandingly in a qualitative way and fail us only when we try to stretch them, as it were, to get quantitative results because the truth of the matter is that for want of better methods we have been obliged to employ these tests as make-shifts and are so accustomed to them that the potential value of more accurate methods is not always fully realized.

The experimental work directed towards finding an improved method led us through all of the possibilities disclosed by the voluminous literature of the subject and ended in the development of an optical instrument which gives rapid, exact and direct measurements of cloudiness or turbidity. The model which was shown at our last meeting has since been greatly improved and refined but I refrain from discussing optics at the present time in order to report an albumin test which has been found exceptionally satisfactory for qualitative as well as quantitative work.

This reagent is really the result of our inability to find a way of coagulating albumin by heat so as to uniformly develop cloudiness proportionate to the amount of albumin in the specimen. There seems to be no escape from the circumstance that the kind and quantity of salts in solution, the $H=ion$ concentration, the specific gravity and a number of other factors, even the thickness of the test tube, the heat of the flame and the proportions of the different globulins to albumin, etc., affect coagulation and influence the quality and quantity of the coagulate. Finding the heat test unfit for quantitative work, numerous and varied albumin precipitants were tried out and by a process of exclusion, a test was developed which exhibits the following characteristics:

1. It is exceedingly simple and rapid.
2. It never reacts with any of the bases or salts found in urine.
3. It does not react with resinous or other foreign substances.
4. It is free of the inaccuracies of layering.
5. It does not require a fixed time for observation.
6. It gives a definite, prompt and reproducible end point.

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7. It shows albumin plainly in specimens which cannot be cleared.
8. It is a shade less sensitive than the heat-salt-acetic acid test.
9. It is specific for serum albumin-globulins and Bence-Jones, and other proteoses.
10. It gives uniformly truthful quantitative clouds or precipitates adapted for optical measurements.

1. The reagent is a single, clear, watery solution which keeps indefinitely. All one has to do is,—mix equal parts of urine and reagent and warm. Even the warmth of a match suffices. Boiling does not spoil the test but is unnecessary and undesirable, because it takes time, affects the reproducibility of the reaction and may vitiate protein differentiation. The use of a small calibre test tube makes the test applicable to minimal amounts of urine and the technique is so simple that a tyro will get satisfactory results with the test.

2. Test made on over 60,000 urines in our own and other laboratories and on urines loaded with uric acid, urates, phosphates, oxalates, urea, creatinine and other urinary constituents greatly in excess have invariably proven negative. Similar reports have been made by previous workers with sulfosalicylic acid and this feature excludes the "pitfalls" which the heat test has for the unwary.

3. Resinous or emulsifying substances have been identified in 14 specimens which gave misleading Heller's and heat reactions but which failed to exhibit the faintest cloud when treated with the new reagent. These findings corroborate Cammidge, MacWilliams and other observers who employ a 20% solution of sulfosalicylic acid as a layer test, as originally recommended by Roche in 1889 and also those who drop a few grains of the dry acid in the urine. The new test was likewise always negative when applied to samples of urine to which oily, gummy and resinous substances were added. Many urines are received in the laboratory which give misleading reactions with Heller's and the heat tests because of substances which are used to preserve the specimens, es-

pecially thymol and excessive amounts of formalin but these will not interfere with the new test, if it be read when warm.

4. All contact or layer tests are merely qualitative because the size and type of the ring depend upon the amount of contact between urine and reagent and other factors. Unavoidable agitation causes inaccuracies which cannot be controlled and tests like Heller's will not yield uniform quantitative deductions in the hands of different technicians. By mixing urine with the reagent this source of error has been eliminated from the new test.

5. For the sake of consistency it is necessary to fix a certain definite time for reading Heller's and many other reactions. This is troublesome, time consuming and subject to inaccuracies due to faulty timing, carelessness, interruptions, etc. The new test does not demand a fixed time for observation but may be read at one's convenience. In the Prudential laboratory technicians mix urines and reagent and leave them stand until one of the chemists finds the time to warm and read the reactions. This feature of the test will be appreciated in laboratories where many specimens are examined.

6. The end point of the reaction given by the new test is attained practically instantly if wanted, and no test has a prompter end point. On the other hand, the test may be set aside for hours, if desired and the end point instantly obtained by warming. If it is later desired to reproduce the reaction in order to check or verify a test which has been made, this may also be done even though the precipitate has fallen, by shaking the test tube and warming. This is a very practicable and unique advantage of the new test.

7. Urines which cannot be cleared may be tested for albumin by comparing the test as done in the regular way with a control made by diluting the urine with an equal part of clear water. The difference in cloudiness between test and control, if any, represents the amount of albumin in specimen.

8. The dilution of the urine with an equal part of clear watery reagent and the fineness of the albumin precipitate as compared with albumin coagulate renders the new test a

shade less delicate than the heat-salt-acetic acid test. The difference in sensitivity, however, is scarcely perceptible to the unaided eye, and those who regard the scant difference in delicacy as a deficiency are reminded that the heat test is not as specific for serum albumin-globulin as the new test and may show hazes caused by other substances than albumin which do not affect the new test.

9. If read when warm the test is practically specific for serum albumin-globulins. When cold, proteoses, protamines and mucin occasion cloudiness. Through the kindness of Dr. H. M. Mosenthal of New York City and Dr. A. H. Sanford of the Mayo Clinic, the reaction with Bence-Jones was found to be very characteristic and "less confusing" than the heat test. In fact, independent observers used the identical expression to describe Bence-Jones precipitate. It looks like "curdled milk" and does not clear up unless the urine is boiled. Nucleo-protein extracted from yeast and thymus glands by Hawkes' technique is not demonstratable in pure solutions or when added to normal urine by the new reagent but as it is known that these nucleo-proteins are not identical with urinary nucleo-protein such experiments are merely suggestive. Of the last 60,000 urines examined in the Prudential laboratory, none contained enough nucleo-protein to show by the new test although Heller's test occasionally suggested its presence and the material of several large clinical institutions has, during the past year, not brought forth, a single specimen containing much nucleo-protein. Evidently, urine containing large amounts of nucleo-protein is rarer than Bence-Jones. Until the sensitivity of the reagent for nucleo-protein can be definitely determined, it will be advisable to test for it separately by treating the diluted urine with acetic acid. Protamins from spermatozoa should be differentiated from secondary proteoses by the microscope because both fall out of solution and develop opalescence when the test cools. As the albumin precipitate is unchanged whether the test be warmed, boiled or cold, it will be seen that the new test is practically specific for albumin-globulins.

10. The clouds or precipitates obtained with heat variously modified, with Heller's, Esbach's, Tsuchiya's and numerous other albumin tests have been checked up by two simple but effective controls for quantitative accuracy, uniformity of reproduction, and specificity as regards albumin-globulins. For one control, 8 or 10 clear albumin-free urines exhibiting the widest differences in composition, H-ion concentration and specific gravity were selected (usually between 1002 and 1040). To equal parts of these and of distilled water exactly the same volume of some albuminous specimen of urine was then added and the test in question applied. For the other control, the test in question was applied to equal parts of progressive dilutions of a clear urine containing much albumin (1 to 2, to 4, to 8, to 16, to 32, to 64, to 128, etc.).

A priori, it was of course assumed that satisfactory tests should precipitate in the one control, precisely the same amounts, and in the other a set of perfect gradations as determined optically or by weighing, and it was therefore a disappointment to learn that mere inspection sufficed to demonstrate the failure of all of the tests which were tried to meet what was considered a necessary, desirable and not too exacting standard.

Our comparisons, however, confirmed the advantages of sulfosalicylic acid for quantitating albumin in urine as found by Folin, Kober and others who employ it in their tests and a study of our results indicated that inconsistencies were traceable directly to differences in the salts and globulins content, specific gravity and H-ion concentration of urines. By using as much reagent as urine and by loading the sulfosalicylic acid solution with sodium sulfate a "buffer" effect is achieved which not only insures complete precipitation of globulins and a high salt content, but which also fixes the H-ion concentration and raises the specific gravity to a point where the differences encountered in specimens of urine are not appreciable. Incidentally, the equilibrium of the albuminate suspensoid is also improved, and when applied to urines of every conceivable normal and pathological content

the new reagent has never failed to meet fully the requirements of both of the controls employed in our work to establish uniformity of reproduction and quantitative accuracy of precipitation.

A liter of reagent is made by dissolving 200 grams of sodium sulfate (crystals) in 700 or 800 cubic centimeters of distilled water. After cooling down to about 38° C., 50 grams of sulfosalicylic acid are dissolved by stirring, and without heating, and enough water added to make one liter. The reagent is not sensitive to light and keeps indefinitely.

The test for albumin is performed as follows:

In a conveniently sized test tube (5 x 1½ in.) mix thoroughly equal parts of urine and reagent. Inspect and warm. Precipitation, if apparent in the warm test, is due to serum albumin-globulins. Opalescence developing on cooling may be due to protamins from spermatozoa or proteoses which the microscope will distinguish. A "curdled milk" precipitate which does not clear unless it is boiled characterizes Bence-Jones. Precipitation is always truthfully representative of the quantity of protein present in the specimen.

Dr. McMahon—Dr. Blakely will lead in the discussion of Dr. Exton's papers.

Dr. Blakely—Mr. President and Gentlemen—Dr. Exton was kind enough to send up to us four or five weeks ago, some of his solution and he asked us to try it out.

He said one thing just now which interested me and that is, that we are all accepting cases with albumin and we realize that we are because the great majority of Examiners, by and large, are not trained nor will they train themselves, to detect the smallest amounts of albumin. When it comes to work done at the Home Office laboratory, it is different. Let me say that, so far as we have gone, we have tried this test qualitatively only, not quantitatively, and we have found that it runs parallel with the cold nitric acid test and with the heat test. We have not found any errors.

We must confess to a prejudice in favor of the nitric acid test, but our chemist, Dr. Hills, has been making that for nearly fifty years continuously day by day, and he has a skill which comparatively few men reach.

One argument against the nitric acid test in the smallest amounts detectable, is the length of time required. There again the average Examiner will not take the time required to do the test properly.

This test of Dr. Exton's seems to have the very great advantage of being quick and quickly decisive, and it is something which any man can train himself to make.

I believe we will get perfectly satisfactory results if we confine ourselves to any one of the standard tests. What we want is a test which in the hands of the majority of men will prove simple and accurate.

I am saying nothing whatever about the preparation of the standard solution. That, I assume, is perfectly simple.

There seem to be no flaws in the technique. It is reduced to the simplest terms and it is certainly satisfactory in the clear-cut results and in the short length of time that is required. I hope that a longer trial will prove that there are no disadvantages and that it is satisfactory both qualitatively and quantitatively, and that we shall have something that we can recommend with assurance to any Examiner who is worthy of undertaking examinations for Life Insurance.

Dr. Pauli—Mr. President and Gentlemen—I did not know that I was to be called on to discuss this paper, but we have had some of the solution in our laboratory and have been working with it, and I can verify the reports made by Dr. Exton. We find it does work beautifully and gives accurate results with the dilute quantities of albumin, and the only point that puzzles us with the reaction is what we are going to call the "dead line." You get the gradation of clouds. You get your one, two, three or four reaction, then you will have to decide whether you are going to call that a positive or a negative reaction.

The mere fact that there are so many reactions for albumin

shows that no reaction yet has been satisfactory, especially from a laboratory standpoint.

I was very much interested in his report of the nitrogen-sugar ratio, because I believe Dr. Exton has something there that may get us away from the glucose test. If we can determine whether we are dealing with a diabetic and if this test will give us that information, we have made splendid progress in our urinalysis work.

Dr. Bradshaw—We started four or five months ago, sending interesting cases to Dr. Lyle's laboratory. The only specimens we sent were those that had these pseudo-reactions, or cases which we suspected were under treatment, or those with long sugar records in which no sugar was found in the urine. At times we split the specimen, saved it for a day or two, and sent it up a little later under another name. The reports checked up every time with what we considered the case to be clinically. A diabetic that we suspected was under treatment was picked out every time. The report was—"This man is a diabetic" although he had no sugar in his urine at all, and the cases with these pseudo-reactions he was very positive in saying that they were not diabetics. We recently had a very interesting case which involved quite a lot of insurance. A man who in 1918 showed .1% was examined for \$100,000 and was declined at that time. He went to a very good clinician who told him that he did not have diabetes; he was also seen by Dr. Joslin who did not think he was a diabetic. He was at that time in a good deal of financial difficulty connected with his business, and he was frequently going back and forth to Baltimore because of his wife's illness, and was altogether having a good deal of trouble. Once or twice he had a sugar reduction, but never enough to give a quantitative test. His business is now straightened out, his wife is better, he is happy in his home again, and is again leading a normal life. His physician was very confident in saying that he did not believe him a diabetic. We got several specimens and finally we sent one to Dr. Lyle, and the report came back that if this was a young man he might be a diabetic; if he was a man over forty he had a very

slight sugar intolerance but was not a diabetic and probably never would be a diabetic. That was a great help to us in this case.

I am sorry I did not hear Dr. Exton's paper. I think there is a good deal in this method of examination for us in Life Insurance, and I would like to see the method thoroughly tried out.

Dr. Fisher—I will not take up your time here, but I have something which may interest you. We have carried down to 1915 the mortality of the Northwestern in cases of Habits in the Use of Stimulants.

On motion of Dr. Rogers, duly seconded, it was ordered that this study of Dr. Fisher's be printed in the transactions.

DR. FISHER'S REPORT ON THE NORTHWESTERN'S
EXPERIENCE WITH RESPECT TO HABITS OF
INSURED PERSONS IN THE USE OF ALCOHOLIC
STIMULANTS—ISSUES 1901-1908

Dr. Fisher—The report in the Transactions of this Association for the years 1917 to 1918 will be found upon pages 208-213, Mortality Experience of the Northwestern Mutual Life Insurance Company with Respect to Habits of Insured Persons in the Use of Alcoholic Stimulants as Compared with Total Abstainers, covering the years 1885-1900, both inclusive.

It will be recalled that the mortality was computed to the policy anniversary in the year 1915, by the Old American Table, classified as to "A—Total Abstainers," "B—Moderate Users of either Malt, Vinous or Spirituous Beverages," "C—Regular Beer Drinkers," and "D—Regular Spirit Drinkers." The data was divided into those below age 40 and ages 40 and above. In all classes the mortality was found to be higher at ages 40 and above, than at ages below 40, by the above Table.

The data submitted herewith covers issues 1901-1908, both

MORTALITY EXPERIENCE OF THE NORTHWESTERN MUTUAL LIFE INSURANCE COMPANY BY
AGES AT ENTRY, AMOUNTS, AND POLICY YEARS, CLASSIFIED WITH REFERENCE TO USE OF
INTOXICANTS. A. M. SELECT AND ULTIMATE TABLE

ISSUES 1901-1908, INCLUSIVE, TO ANNIVERSARY IN 1915

A—Total Abstainers

Entry Age	FIRST 5 YEARS		AFTER 5 YEARS		Poles.	Deaths	TOTAL		Per Cent.
	Actual	Tabular	Actual	Tabular			Actual	Tabular	
16-29	1,121,300	1,437,270	1,060,700	1,575,471	61,944	1,477	2,182,000	3,012,741	72.4
30-39	1,035,600	1,602,849	1,361,400	2,229,899	41,500	1,226	2,397,000	3,832,748	62.5
40-49	1,445,500	1,837,065	2,049,700	2,997,763	21,607	1,273	3,495,200	4,834,828	72.3
50-59	1,054,400	1,365,336	1,604,000	2,308,256	7,207	904	2,658,400	3,673,592	72.4
60 & Up	39,000	127,995	189,000	263,835	391	87	228,000	391,830	58.2
Total	4,695,800	6,370,515	6,264,800	9,375,224	132,649	4,967	10,960,600	15,745,739	69.6
	73.7%		66.8%				69.6%		

B—Moderate Users

16-29	838,500	1,098,231	1,028,800	1,212,979	34,695	870	1,867,300	2,311,210	80.8
30-39	1,770,800	2,500,243	2,882,800	3,422,414	42,457	1,482	4,653,600	5,922,657	78.6
40-49	2,516,000	3,217,890	3,581,400	5,137,275	24,807	1,573	6,097,400	8,355,165	73.0
50-59	1,850,500	2,293,648	3,040,400	3,825,015	7,740	1,102	4,890,900	6,118,663	79.9
60 & Up	204,300	216,659	331,600	328,467	437	107	536,100	545,126	98.3
Total	7,180,300	9,326,671	10,865,000	13,926,150	110,136	5,224	18,045,300	23,252,821	77.6
	77.0%		78.0%				77.6%		

C—Regular Beer Drinkers

16-29	141,000	132,114	130,700	141,060	4,807	126	271,700	273,174	99.5
30-39	248,000	312,731	398,800	417,853	6,324	254	656,800	730,584	89.9
40-49	317,500	339,633	404,300	549,312	3,292	232	721,800	888,345	81.3
50-59	197,500	204,783	344,400	333,722	931	141	541,900	538,505	100.6
60 & Up	27,500	17,059	27,500	17,040	58	14	55,000	34,099	161.3
Total	941,500	1,005,720	1,305,700	1,458,987	15,412	767	2,247,200	2,464,707	91.2

91.2%

D—Regular Spirit Drinkers

16-29	12,000	17,585	22,100	16,352	342	8	34,100	33,937	100.5
30-39	223,200	105,532	190,300	124,662	1,235	97	413,500	230,194	179.6
40-49	276,500	198,444	386,000	280,802	1,116	89	662,500	479,246	138.2
50-59	281,500	194,165	190,500	288,610	482	88	472,000	482,775	97.8
60 & Up	18,000	12,300	1,000	19,321	31	7	19,000	31,621	60.1
Total	811,200	528,026	789,900	729,747	3,206	289	1,601,100	1,257,773	127.3

127.3%

All Classes

16-29	2,112,800	2,685,200	2,242,300	2,945,862	101,788	2,481	4,355,100	5,631,062	77.3
30-39	3,287,600	4,521,355	4,833,300	6,194,828	91,516	3,059	8,120,900	10,716,183	75.8
40-49	4,555,500	5,592,432	6,421,400	8,965,152	50,822	3,167	10,976,900	14,557,584	75.4
50-59	3,383,900	4,957,932	5,179,300	6,755,603	16,300	2,325	8,563,200	10,813,535	79.2
60 & Up	289,000	374,013	549,100	628,663	917	215	838,100	1,002,676	83.6
Total	13,628,800	17,230,932	19,225,400	25,490,108	261,403	11,247	32,854,200	42,721,040	76.9

76.9%

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inclusive, and carried to anniversary in the year 1915, by the American Men Select and Ultimate Table. It will be seen that there is practically no difference in the percentage of mortality at ages below 40 and at ages 40 and above. In the Total Abstainers and Moderate Users as well as in the classes of Beer Drinkers and Spirit Drinkers there is some fluctuation, which is no doubt due to the comparatively smaller number in those two classes. Combining all classes, the mortality is practically the same at all group ages.

Your attention is called to the specific rules which govern the classification as recorded by a member of the Home Office Medical Staff at the time of the approval of the application and after full investigation of all cases in which there was any doubt as to the statements of the applicant upon the application blank. The Company has been especially careful in its selection of applicants with respect to the use of stimulants. During the period covered by the above data, we secured Inspection Reports in practically all cases.

RULES GOVERNING SELECTION

A. *Total Abstainers.*

B. *Moderate Users of Either Malt, Vinous or Spirituous Beverages.*

Wine drinkers only, up to four glasses daily.

Beer or ale, not daily or more than 3 in any one day at the most.

Porter or strong ale not daily or more than 2 in any one day at the most.

Light wine " " " " " 4 " " " " " "

Strong wine " " " " " 3 " " " " " "

Spirituous Liquor " " " " " 2 " " " " " "

Daily—1 glass of beer or wine or whiskey.

May take more than one or all kinds on same day, but no one kind more than above.

C. *Regular Beer Drinkers.*

One who uses four or more glasses of beer or ale in any one day at most, or 5 or more in a week, or a daily practice of 1 or more. Include where wine or whiskey is also used moderately *but not* enough for class "D."

No wine only cases, in this class.

Two glasses of beer or wine a day.

Beer and wine at meals, moderately.

Porter or strong ale daily, or 3 or more in any 1 day at the most.

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D. *Regular Spirit Drinkers.*

One who uses all kinds but not entitled to Class "B."

Wine daily, 5 light, 4 strong, or more.

Spirituous liquors daily, or 3 or more in any 1 day at the most.

Average two a day, moderately, about two a day.

Two glasses of beer or wine or whiskey, a day.

Dr. McMahon—Dr. Henry Wireman Cook will describe to us at this time a Visual Albuminuria Guide.

A VISUAL ALBUMINURIA GUIDE

BY HENRY WIREMAN COOK, M.D.

Vice-President and Medical Director, Northwestern National Life Insurance Co., Minneapolis, Minn.

I am taking the liberty of showing a suggestion for roughly determining the amount of albumin present in a urinary specimen and more especially to offer a comparable and relatively constant nomenclature for varying degrees of albuminuria where exact quantitative tests are not possible or convenient. In life insurance examinations the need for such a method is most imperative, as quantitative estimates are out of the question and as the vague terms in common use, such as trace, moderate amount, etc., vary so with the opinion of the observer and the delicacy of his technique that they have little accurate value. In a large proportion of routine clinical and hospital work, this same difficulty is met with, and this visual guide is offered as an assistance in standardizing our nomenclature in at least approximate relation to definite quantitative amounts.

The tests selected as standard are the Heat and Acid and Heller's because they are the most widely known and most easily done, and because Heller's especially well lends to quantitative estimates. In our laboratory we use Ulrich's

ring modification of the Heat and Acid as the initial test, as it is more delicate than either the Heat and Acid as ordinarily applied, or Heller's, but as it is no so generally known, we have not reproduced it in our chart.

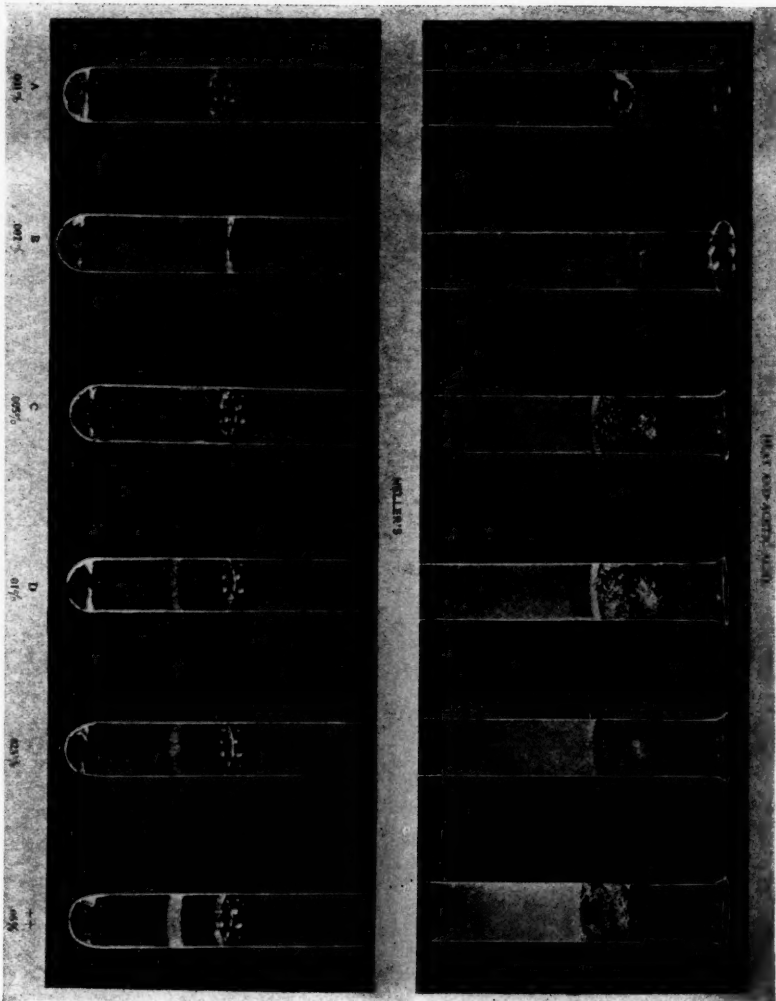
The tests shown in the chart are photographic reproductions of actual tests made with known solutions of an albumin from a case of nephritis. Previous attempts to standardize notations of degrees of albuminuria have depended upon verbal descriptions which attempt to measure the width of the albumin ring or picture its density. There is a well recognized relation between the width and density of the cloud and the amount of albumin. But descriptions which depend upon attempts to measure the ring in fractions of an inch or to gauge density by ability to read printing through the cloud are obviously inaccurate. It is believed that by presenting visually the coagulated albumin as it appears in actual tests, with careful attention to standardizing the method of making the tests, results will be obtained approximately uniform. This has been the experience in our own laboratory and in the laboratories of a number of our examiners who are using the visual chart.

As it is essential that the tests should be made under uniform conditions, these are here detailed:

1. The test tube must be scrupulously clean.
 2. The test is to be made at the window in a bright light or with strong artificial light against a black background. A test made at the middle or back of the room, where, in a doctor's office, the sink is frequently placed, and held at that distance towards the window, will only show the larger amounts of albumin.
 3. The reading should be made after one minute and within three minutes. Before one minute the smaller traces will not appear with Heller's test, and after three minutes the amount is exaggerated by the gradual spread of the ring upward.
- Heat and Acid.* The clean test tube is filled three-quarters full of the filtered urine, which, if neutral or alkaline, should be acidulated by adding a few drops of 3% acetic acid. The

VISUAL ALBUMINURIA GUIDE

HEAT AND ACETIC ACID TEST



HEAT AND ACETIC ACID TEST. A clean test-tube is filled three-fourths full of the clear neutral or faintly acid filtered urine and heated at the top to a boil, the tube being held by its lower end. If a cloud is produced, it can be easily recognized by comparing the upper with the lower half of the tube while holding the tube against a black background. A cloud may be due to albumin, or to phosphates or carbonates. If the cloud is albumin it will rather increase, whereas if due to phosphates or carbonates it will disappear on the addition of acid.

HERRING'S TEST. Place in a clean test-tube about one inch of concentrated nitric acid; then incline the tube almost horizontally; from a pipette allow an equal volume of urine to flow slowly down the side of the tube, carefully overlapping the acid. Wait three minutes. If albumin is present, by the use of a black background a zone will be seen just above the point of contact of the urine and the acid, the intensity and width of the cloud depending on the amount of albumin present.



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upper portion of the tube, held at the lower end, should be heated until the urine boils. If this is held against a black background in a strong direct light the coagulated albumin will be seen in a cloud of varying thickness depending on the amount present. It can be estimated by comparison with the chart.

Heller's: About an inch of the clean test tube is filled with concentrated nitric acid. The tube is then held in an almost horizontal position and the filtered urine allowed to flow *very slowly* down the tube to overlay the acid. If the urine and acid are mixed so that the ring of contact is not even and distinct the test should be done over.

At the point of contact, albumin, if present in an amount greater than 1 part to 1000, will appear as a coagulated white ring of varying width and density, depending on its concentration. The amount of albumin present will also determine how soon the ring becomes visible. In concentration greater than 1 to 500 the ring appears immediately. In amounts between 1 to 1000 and 1 to 500 a very faint ring appears in from one-half to one minute.

Dr. McMahon—It is now my great pleasure to introduce to you the incoming President, Dr. Grosvenor. I wish only to express the hope that he will have extended to him the same kindness, courtesy and consideration which has been shown to me. I am deeply grateful to the members of the Association for the very pleasant path they have made it possible for me to tread. I think on the whole the meeting has been a success, and it has been due to the spirit of the members and to their willingness to cooperate in bringing about a successful meeting. It gives me great pleasure, gentlemen, to introduce to you the incoming President of the Association of Life Insurance Medical Directors, Dr. Frank L. Grosvenor.

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Dr. Grosvenor was greeted with applause as he took the chair.

Dr. Grosvenor—I am deeply appreciative, Gentlemen of the Association, and I have nothing to say except that I anticipate the same cooperation and courtesy and interest in the coming year and in the coming meeting which Dr. McMahon has received from the members of the Association.

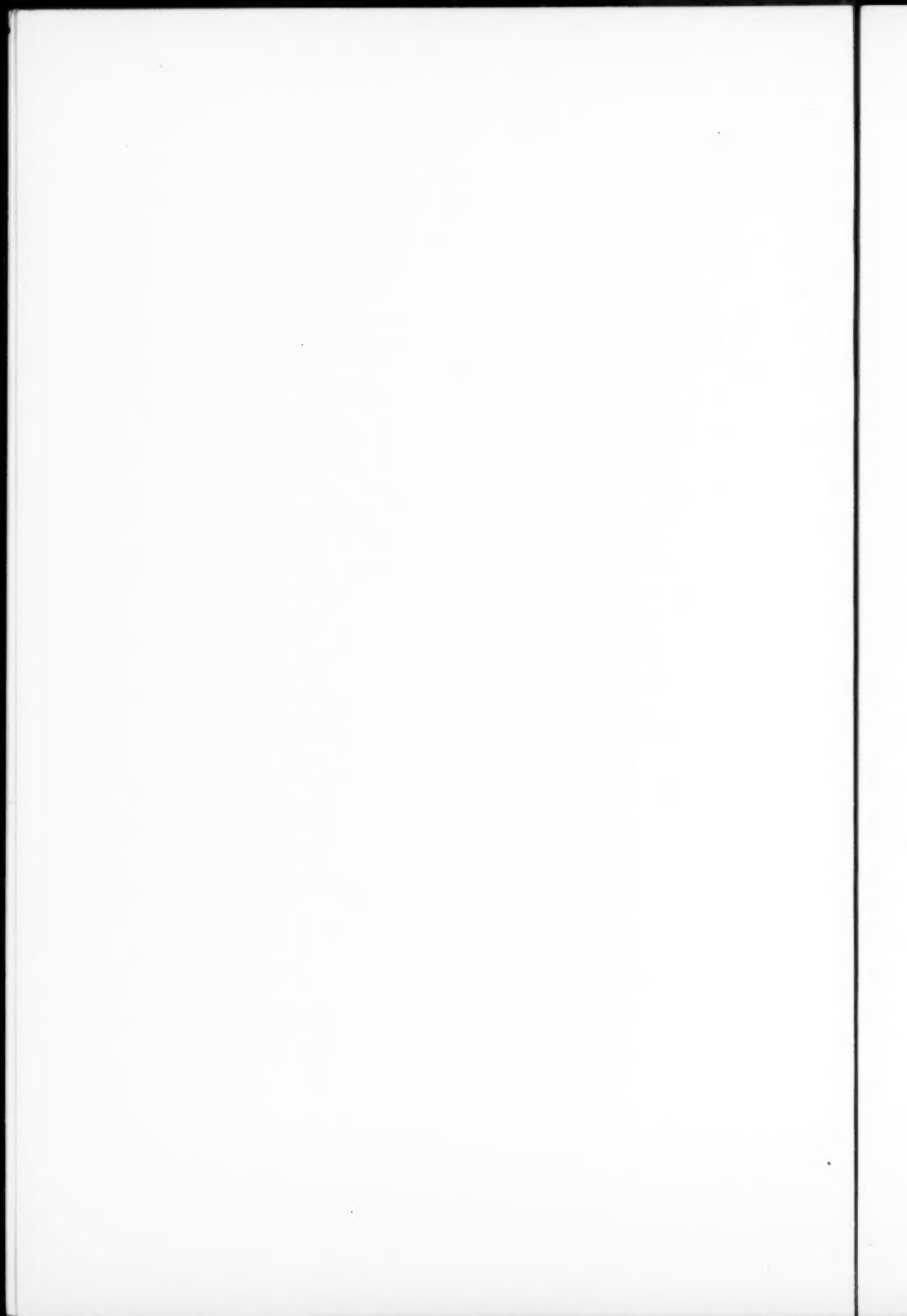
Dr. Porter—Gentlemen—Before we close, I move that we, as members of this Association, extend to President Kingsley and the other officers of the New York Life Insurance Company, our hearty thanks and appreciation of the cordiality, courtesy and hospitality which they have extended to us during this meeting. The motion was seconded and carried by a rising vote.

There being no further business, the meeting adjourned sine die.

The Annual Dinner of the Association of Life Insurance Medical Directors was held on the evening of Thursday, November 2, 1922, at the Manhattan Club, New York City, N. Y. The following members, delegates and guests were present:

John L. Adams, H. B. Anderson, T. D. Archibald, H. A. Baker, A. W. Balch, Wm. B. Bartlett, J. T. J. Battle, W. W. Beckett, Chas. D. Bennett, David N. Blakely, Wm. M. Bradshaw, Chester T. Brown, Wm. H. Browne, T. W. Burrows, J. T. Cabaniss, Frank H. Carber, L. D. Chapin, Chas. L. Christiernin, G. E. Crawford, John N. Coolidge, Robert M. Daley, Edwin G. Dewis, W. E. Dickerman, Wm. W. Dins-

more, Percy G. Drake, H. K. Dunham, Edwin W. Dwight, O. M. Eakins, Z. H. Ellis, C. H. English, Wm. G. Exton, Paul Fitzgerald, R. A. Fraser, H. M. Frost, S. W. Gadd, F. Irving Ganot, Wm. S. Gardner, A. H. Griswold, F. L. Grosvenor, Geo. C. Hall, Wm. F. Hamilton, Wm. J. Hammer, Frank H. Harnden, Whitfield Harral, C. L. Harrison, A. B. Hobbs, J. F. Honsberger, Ross Huston, Wm. G. Hutchinson, Lefferts Hutton, Arthur Hunter, Chas. B. Irwin, Walter A. Jaquith, F. L. B. Jenney, A. O. Jimenis, Albert E. Johann, Geo. E. Kanouse, R. J. Kissock, A. S. Knight, W. W. Knight, M. M. Lairy, E. H. Lines, R. L. Lounsberry, Charles N. McCloud, L. F. McKenzie, T. F. McMahon, Paul Mazzuri, Samuel W. Means, George L. Megargee, Jas. H. North, Martin I. Olsen, Wm. O. Pauli, J. A. Patton, Wm. A. Peterson, J. S. Phelps, Charles B. Piper, Jos. E. Pollard, Wm. Evelyn Porter, James T. Priestley, Thomas H. Rockwell, Oscar H. Rogers, Eugene F. Rowley, C. E. Schilling, Samuel B. Scholz, Jr., A. L. Sherrill, James M. Smith, Malcolm K. Smith, Morton Snow, Marion Souchon, Howard B. Speer, H. F. Starr, Carl Stutsman, P. E. Tiemann, Harry Toulmin, J. P. Turner, C. A. Van Dervoort, Euen Van Kleeck, Wm. H. E. Wehner, Faneuil S. Weisse, F. C. Wells, Fred L. Wells, C. F. S. Whitney, Thomas H. Willard.



LIST OF MEMBERS OF THE ASSOCIATION OF LIFE INSURANCE MEDICAL DIRECTORS

Frederick R. Abbe.	Columbian National, Boston, Mass.
John L. Adams, M.D.	Metropolitan, New York, N. Y.
Edwin H. Allen, M.D.	John Hancock, Boston, Mass.
Charles D. Alton, M.D.	Connecticut Mutual, Hart- ford, Conn.
John W. Ames, M.D.	Capitol Life of Colorado, Denver, Colo.
Harry B. Anderson, M.D.	Imperial Life, Toronto, Ont.
Thomas D. Archibald, M.D.	North American, Toronto, Ont.
William Armstrong, M.D.	Connecticut General, Hart- ford, Conn.
Alfred W. Balch, M.D.	Metropolitan, New York, N. Y.
William B. Bartlett, M.D.	John Hancock, Boston, Mass.
John T. J. Battle, M.D.	Jefferson Standard, Greens- boro, N. C.
Wesley W. Beckett, M.D.	Pacific Mutual, Los Angeles, Calif.
Charles D. Bennett, M.D.	Mutual Benefit, Newark, N. J.
Thomas W. Bickerton, M.D.	New York Life, New York, N. Y.
Edward B. Bigelow, M.D.	State Mutual, Worcester, Mass.

List of Members

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Cecil C. Birchard, M.D.	Sun Life, Montreal, Quebec.
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David N. Blakely, M.D.	New England Mutual, Boston, Mass.
Robert J. Blanchard, M.D.	Great West, Winnipeg, Manitoba.
William M. Bradshaw, M.D.	Mutual, New York, N. Y.
Frederick G. Brathwaite, M.D.	Equitable, New York, N. Y.
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Chester T. Brown, M.D.	Prudential, Newark, N. J.
William H. Browne, M.D.	American Life, Detroit, Mich.
Chauncey R. Burr, M.D.	Metropolitan Life, New York, N. Y.
Joseph T. Cabaniss, M.D.	Travelers, Hartford, Conn.
Frank H. Carber, M.D.	Mutual, New York, N. Y.
Frank W. Chapin, M.D.	Home Life, New York, N. Y.
Laurence D. Chapin, M.D.	Mass. Mutual, Springfield, Mass.
John P. Chapman, M.D.	Penn Mutual, Philadelphia, Pa.
Charles L. Christiernin, M.D.	Metropolitan, New York, N. Y.
Charles P. Clark, M.D.	Mutual Benefit, Newark, N. J.
Henry Colt, M.D.	Berkshire, Pittsfield, Mass.
Edwin A. Colton, M.D.	National, Montpelier, Vt.
Henry W. Cook, M.D.	Northwestern Nat., Minneapolis, Minn.
John N. Coolidge, M.D.	Metropolitan, New York, N. Y.
Robert M. Daley, M.D.	Equitable, New York, N. Y.
Edwin Grafing Dewis, M.D.	Prudential, Newark, N. J.
Wilton E. Dickerman, M.D.	Ætna, Hartford, Conn.
Henry K. Dillard, M.D.	Penn Mutual, Philadelphia, Pa.

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Percy G. Drake, M.D.	Travelers, Hartford, Conn.
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William G. Exton, M.D.	Prudential, Newark, N. J.
John Ferguson, M.D.	Excelsior, Toronto, Ont.
John W. Fisher, M.D.	Northwestern Mutual, Milwaukee, Wis.
Paul Fitzgerald, M.D.	Prudential, Newark, N. J.
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Samuel W. Gadd, M.D.	Philadelphia Life, Philadelphia, Pa.
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William S. Gardner, M.D.	Mutual Life, New York, N. Y.
Donald M. Gedge, M.D.	Metropolitan, New York, N. Y.
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Fred Le M. Grasett, M.D.	Canada Life, Toronto, Ont.
Arthur H. Griswold, M.D.	Phoenix Mutual, Hartford, Conn.
Frank L. Grosvenor, M.D.	Travelers, Hartford, Conn.
Ignatius Haines, M.D.	John Hancock, Boston, Mass.
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William F. Hamilton, M.D.	Sun Life, Montreal, Que.

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List of Members

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Carleton B. McCulloch, M.D.	State Life, Indianapolis, Ind.
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Samuel W. Means, M.D.	Metropolitan, New York, N. Y.
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William Evelyn Porter, M.D.	Mutual, New York, N. Y.
Albert T. Post, M.D.	Equitable, New York, N. Y.

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H. Crawford Scadding, M.D.	Canada Life, Toronto, Ont.
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Brandreth Symonds, M.D.	Mutual Life, New York, N. Y.
William Thorndike, M.D.	Northwestern Mutual, Milwaukee, Wis.
Paul E. Tiemann, M.D.	New York Life, New York, N. Y.

Harry Toulmin, M.D.	Penn Mutual, Philadelphia, Pa.
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William H. E. Wehner	Fidelity Mutual, Philadelphia, Pa.
Faneuil S. Weisse, M.D.	Mutual, New York, N. Y.
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Franklin C. Wells, M.D.	Equitable, New York, N. Y.
Fred L. Wells, M.D.	Equitable of Iowa, Des Moines, Iowa.
David E. W. Wenstrand, M.D.	Northwestern Mutual, Milwaukee, Wis.
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Chester F. S. Whitney, M.D.	Home Life, New York, N. Y.
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Gordon Wilson, M.D.	Maryland Life, Baltimore, Md.
McLeod C. Wilson, M.D.	Travelers, Hartford, Conn.

List of Members

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Harry P. Woley, M.D.

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N. Y.

Glenn Wood, M.D.

Illinois Life, Chicago, Ill.

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Granville M. White, M.D.

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John W. Brannan, M.D.

New York, N. Y.

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American Life, Detroit, Mich.	W. H. Browne, M.D.
Atlantic Life, Richmond, Va.	F. P. Righter, M.D.
Bankers Life, Des Moines, Iowa	{ Ross Huston, M.D. Albert E. Johann, M.D.
Bankers Reserve Life, Omaha, Neb.	
Berkshire Life, Pittsfield, Mass.	{ Henry Colt, M.D. B. W. Paddock, M.D.
Canada Life Assurance Co., Toronto, Ont., Can.	{ F. L. M. Grasett, M.D. H. C. Scadding, M.D. G. S. Strathy, M.D.
Capitol Life of Colorado, Denver, Colo.	John W. Amessee, M.D.

Companies and Representatives 293

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Connecticut Mutual Life, Hartford, Conn.	{ C. D. Alton, M.D. J. B. Hall, M.D. H. A. Martelle, M.D.
Equitable Life Assur. Soc., New York, N. Y.	{ A. W. Billing, M.D. F. G. Brathwaite, M.D. R. M. Daley, M.D. Arthur Geiringer, M.D. A. T. Post, M.D. T. H. Rockwell, M.D. A. H. Sherrill, M.D. F. C. Wells, M.D.
Equitable Life of Iowa, Des Moines, Iowa	F. L. Wells, M.D.
Excelsior Life, Toronto, Ont., Can.	John Ferguson, M.D.
Fidelity Mutual Life, Philadelphia, Pa.	{ C. A. VanDervoort, M.D. W. H. E. Wehner, M.D.
Franklin Life, Springfield, Ill.	O. F. Maxon, M.D.
Guardian Life, New York, N. Y.	C. B. Piper, M.D.

294 Companies and Representatives

Great West Life Assur. Co., Winnipeg, Man., Can.	R. J. Blanchard, M.D.
Gresham Life, Montreal, Que., Can.	H. A. Lafleur, M.D.
Home Life, New York, N. Y.	{ F. W. Chapin, M.D. C. F. S. Whitney, M.D.
Illinois Life, Chicago, Ill.	Glenn Wood, M.D.
Imperial Life Assur. Co., Toronto, Ont., Can.	H. B. Anderson, M.D.
Jefferson Standard Life, Greensboro, N.C.	{ J. T. J. Battle, M.D. J. P. Turner, M.D.
John Hancock Mutual Life, Boston, Mass.	{ E. H. Allen, M.D. W. B. Bartlett, M.D. I. Haines, M.D. E. B. Kellogg, M.D.
Life Ins. Co. of Virginia, Richmond, Va.	G. C. Hall, M.D.
Lincoln National Life, Fort Wayne, Ind.	C. H. English, M.D.
London & Scot. Assur. Ass'n., Ltd. Montreal, Que., Can.	A. H. Gordon, M.D.
London Life, London, Ont., Can.	Angus Graham, M.D.
Manhattan Life, New York, N. Y.	Z. Taylor Emery, M.D.

Companies and Representatives 295

Manufacturers Life, Toronto, Ont., Can.	T. F. McMahon, M.D.
Maryland Life, Baltimore, Md.	Gordon Wilson, M.D.
Massachusetts Mutual Life, Springfield, Mass.	{ L. D. Chapin, M.D. R. B. Ober, M.D. S. B. Scholz, Jr., M.D. Morton Snow, M.D. G. S. Stebbins, M.D.
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Mutual Life Assurance Co., Waterloo, Ont., Can.	{ J. F. Honsberger, M.D. J. M. Livingston, M.D.
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National Life, Montpelier, Vt.	{ A. B. Bisbee, M.D. E. A. Colton, M.D.
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Companies and Representatives 297

New York Life, New York, N. Y.	<div style="display: flex; align-items: center;"> <div style="font-size: 3em; margin-right: 5px;">{</div> <div> T. W. Bickerton, M.D. R. A. Fraser, M.D. W. J. Hammer, M.D. C. L. Harrison, M.D. A. B. Hobbs, M.D. M. L. King, M.D. E. H. Lines, M.D. Paul Mazzuri, M.D. J. H. North, M.D. O. H. Rogers, M.D. P. E. Tiemann, M.D. H. P. Woley, M.D. </div> </div>
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North American Life, Toronto, Ont., Can.	T. D. Archibald, M.D.
Northwestern Mutual Life, Milwaukee, Wis.	<div style="display: flex; align-items: center;"> <div style="font-size: 3em; margin-right: 5px;">{</div> <div> J. W. Fisher, M.D. G. A. Harlow, M.D. William Thorndike, M.D. D. E. W. Wenstrand, M.D. </div> </div>
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Pacific Mutual Life, Los Angeles, Calif.	W. W. Beckett, M.D.
Penn Mutual Life, Philadelphia, Pa.	<div style="display: flex; align-items: center;"> <div style="font-size: 3em; margin-right: 5px;">{</div> <div> L. D. Bristol, M.D. J. P. Chapman, M.D. H. K. Dillard, M.D. J. U. Hobach, M.D. J. P. Hutchinson, M.D. Harry Toulmin, M.D. </div> </div>
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Companies and Representatives 299

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Lincoln National Life, Ft. Wayne, Ind.	{ D. M. Shewbrooks, M.D. W. E. Thornton, M.D.

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Union Central Life, Cincinnati, Ohio	{ Euen Van Kleeck, M.D.
	Charles Maertz, M.D.

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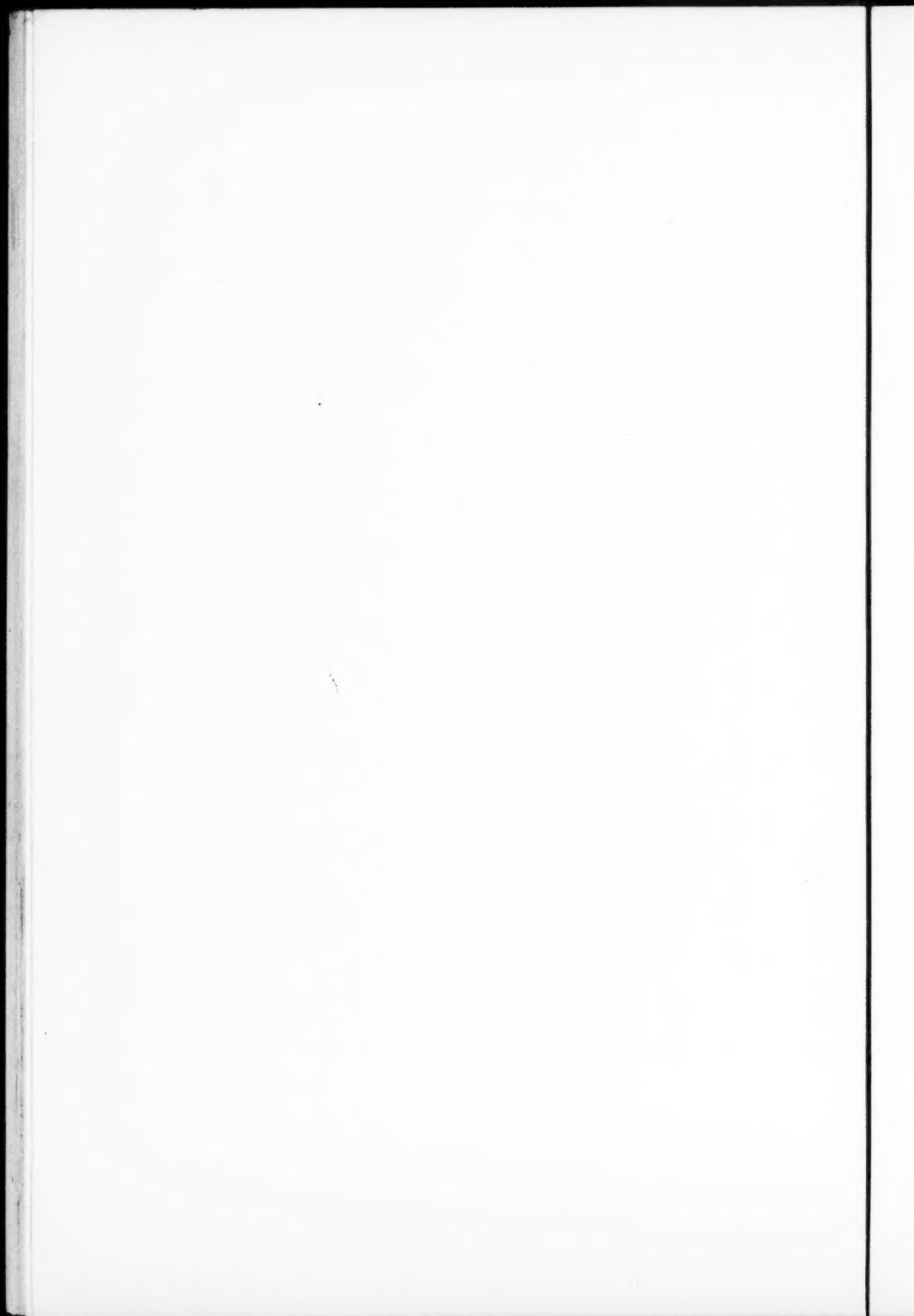
Central Life Insurance Co. of Illinois,
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Occidental Life Insurance Co.,
Los Angeles, Calif.
Southland Life Insurance Co.,
Dallas, Tex.
Southwestern Life Insurance Co.,
Dallas, Tex.
West Coast Life Insurance Co.,
San Francisco, Calif.

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Dr. M. M. Lairy, LaFayette Life Ins. Co., LaFayette, Ind.
Dr. G. E. Crawford, Cedar Rapids Life Insurance Co., Cedar
Rapids, Iowa.
Dr. B. C. Brooke, Montana Life Ins. Co., Helena, Mont.
Dr. Carl Stutsman, Merchants Life Insurance Co., Des
Moines, Iowa.
Dr. W. F. Blackford, Commonwealth Life Insurance Co.,
Louisville, Ky.
Dr. C. E. Schilling, Ohio State Life Ins. Co., Columbus, Ohio.
Dr. T. W. Burrows, Central Life Insurance Co. of Illinois,
Ottawa, Illinois.
Dr. M. L. Turner, Western Life Ins. Co. of Iowa, Des Moines,
Iowa.

ALTERNATES

- Dr. A. J. Giesy, Oregon Life Insurance Co., Portland, Oregon.
Dr. M. T. McCarty, Peoples Life Ins. Co., Frankfort, Ind.
Dr. H. E. Sharrer, Northern States Life Ins. Co., Hammond,
Ind.
Dr. S. C. Stanton, Farmers National Life Ins. Co., Chicago,
Illinois.
Dr. John W. Abbott, Maryland Assurance Corporation, Balti-
more, Maryland.



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James Campbell, M.D.	Hartford, Conn.
Frederick W. Chapin, M.D.	Springfield, Mass.
Ferdinand E. Chatard, M.D.	Baltimore, Md.
Thomas C. Craig, M.D.	New York, N. Y.
Edward Curtis, M.D.	New York, N. Y.
Clark W. Davis, M.D.	Cincinnati, Ohio
William B. Davis, M.D.	Cincinnati, Ohio
Charles A. Devendorf, M.D.	Detroit, Mich.
Frank Donaldson, M.D.	Baltimore, Md.
Thomas A. Foster, M.D.	Portland, Me.
Thomas H. Gage, M.D.	Worcester, Mass.
Walter R. Gillette, M.D.	New York, N. Y.
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